New Orleans Medical
and
Surgical Journal

Vol. 95 JANUARY, 1943 No. 7

THE FUTURE OF THE PATIENT WITH CORONARY OCCLUSION*

CHARLES T. STONE, M. D.†

GALVESTON, TEXAS

Within the past quarter of a century coronary occlusion has become one of the most important clinical entities in the field of internal medicine. The myocardial infarction that results from closure of a coronary artery produces such characteristic symptoms, signs, and electrocardiographic changes that its recognition is comparatively easy, and physicians generally arrive at the diagnosis correctly in a large majority of instances. In fact the clinical findings are so well known that they will receive only passing comment in this presentation.

It is of paramount significance that when an individual sustains a coronary occlusion he is from that moment on confronted with a problem of considerable magnitude, and one which will most probably have an important bearing on the remainder of his life. He may die from the acute phase of the illness, he may be left in a permanently disabled state that in some instances is partial, in others total, or he may recover more or less completely. It is my purpose to attempt to show what the coronary occlusion patient may expect of the future if he is fortunate enough to survive the initial effects of the occlusion and what his resulting status will be.

*Read before the sixty-third annual meeting of the Louisiana State Medical Society, New Orleans, April 29, 1942.

†From the Department of Internal Medicine, The University of Texas Medical School, Galveston, Texas.

Every physician knows that considerable uncertainty exists in his ability to give an accurate prognosis in the case of a patient acutely ill from coronary occlusion. It is not uncommon to see those who are only slightly to moderately ill—both subjectively and objectively—die suddenly and unexpectedly, or from an unpredictable complication after a few hours, a few days, or a few weeks. Others much more gravely ill recover and may be able to lead nearly normal and useful lives for a good many years. Yet there are certain facts which stand out as fairly trustworthy guideposts in the effort to arrive at a correct prognosis.

In a recent publication Shillito, Chamberlain, and Levy[1] have emphasized the highly fatal significance of; first, a rectal temperature above 104 F., second, a leukocyte count of 25,000 or above, and third, a venous pressure over 200 mm. of water. Seven of eight patients in their series of 50 cases of uncomplicated coronary occlusion showing any one of these signs died within 16 days after the occlusion occurred. Only ten of the remaining 42 patients not presenting any of these signs succumbed. A temperature above 104 F., and a leukocyte count in excess of 25,000 speak strongly for a massive myocardial infarct, and it may reasonably be expected that the larger the lesion the less likely is recovery to occur. Venous pressure above 200 mm. of water indicates a high grade congestive heart failure, a factor that is admittedly of unfavorable omen.

Similarly, it is frequently observed that severe shock, marked tachycardia—120 per minute or above—deep cyanosis, pulsus alternans, gallop rhythm, enfeebled heart sounds and profound, and prolonged drop in blood pressure (80 mm. Hg. systolic or

below) especially with a low pulse pressure carry a much more serious outlook than when these findings are absent or minimal. Again it should be emphasized that these signs, together with a rising venous pressure indicate that the degree of circulatory failure, both peripheral and central, is great, and as they occur in increasing severity the prognosis becomes worse. The intensity of the pain alone is not a reliable index to the seriousness of the acute episode.

Certain information of prognostic significance can be obtained from electrocardiograms particularly if they are made at frequent intervals early in the course of the illness. Subsequently they are highly useful in estimating the progress of recovery in the weeks and months after the acute process has subsided. Patients with curves showing bundle branch block and auriculoventricular block have the worst outlook for recovery, but in many such instances the acute phase passes, and restitution to good health follows. In complete heart block with Stokes-Adams seizures the prognosis is particularly unfavorable. In 10 to 15 per cent of the cases the electrocardiograms are non-confirmatory and may be normal or nearly so. Generally, this carries a favorable prognosis. Location of the site of the lesion is indicated by the curves in about two-thirds of the cases. Such studies show that anterior infarctions are somewhat more common than those located in the posterior wall of the heart, but such findings have little if any prognostic significance so far as recovery from the acute attack is concerned. Both groups show about the same immediate mortality. However, the longevity is shorter in those with anterior infarctions since they show a greater tendency to develop congestive heart failure than do posterior lesions.

Several writers, more recently Woods and Barnes,[2] have stressed the importance of frequent extrasystoles of ventricular origin. When ventricular premature contractions occurred as frequently as one in every two to ten heart beats, 82 per cent of their patients died within the acute period. The assumption is that the infarcted area constitutes a highly irritable focus which initiates premature contractions. When these are frequent it is considered probable that they in turn may inaugurate ventricular fibrillation, an important cause of sudden death in coronary occlusion. Therefore, it is felt that frequent ventricular extrasystoles demand that oral quinidine therapy be promptly instituted and energetically carried out.

A small number of fatalities results from complications, chiefly embolic. Cerebral, renal, mesenteric, splenic emboli, and those to the extremities arise usually from mural thrombi on the wall of the left ventricle. Pulmonary emboli most frequently come from thrombi in the femoral and iliac veins. This occurred in 10 per cent of Woods and Barnes[2] cases. Pneumonia and other intercurrent infections account for another small percentage of deaths.

INCIDENCE

Men have coronary occlusion much more frequently than women in ratios variously reported from 3 to 7:1, and yet in almost every statistical study the death rate for women greatly exceeds that for men. It is a well known fact that the older the individual the less likely is recovery from coronary occlusion. Thus in age groups below 40 considerably less than 30 per cent fail to survive the acute phase; between 40 and 70 about 30 per cent die. In those above 70 the rate of mortality is 50 per cent or more. Since women tend to develop coronary occlusion at an age several years later than men some of the increased severity of the prognosis for immediate recovery in women may be explained on that basis. Silberberg[3] suggests that increased frequency of hypertension in females may be a factor, but the fact remains that we do not have an entirely satisfactory answer to this question.

PROGNOSIS

In attempting to gauge the prognosis on the basis of age and sex, however, no very reliable criteria are available. It might be said that regardless of age or sex the important points to look to for guidance are: the rectal temperature, the leukocyte count, the venous pressure, the degree of shock as measured by the fall in blood pressure, the

lowness of the pulse pressure, the degree of cyanosis, the presence of marked tachycardia, the occurrence of serious disturbances of cardiac rhythm, certain electrocardiographic changes (already mentioned), and a progressive, unchecked congestive heart failure and peripheral circulatory failure. These signs singly or in various combinations bode ill for either sex and at all ages.

Practically all writers on the subject of the immediate prognosis in coronary thrombosis comment upon the extreme difficulty involved in giving precise estimates in given cases. In that opinion I am in complete agreement, but I do believe that a careful appraisal of each case, bearing in mind the facts cited in the foregoing paragraphs and combined with good clinical judgment will enable the physician to make the smallest possible number of errors.

MORTALITY

In an effort to determine the immediate fatality rate following coronary occlusion the statistics from a number of widely scattered centers have been collected, and the results tabulated in table 1.

TABLE 1

FATALITY RATE

Author	No. of cases	Death within 4 to 6 weeks No.	Per cent
Connor and Holt[4]	287	46	16.2
Silberberg[3]	282	63	22.3
Clark[5]	19	9	47.4
Levine[6]	143	76	53
Howard[7]	165	40	24.24
Master, Jaffe, and Dack[8]	243	20	8
Willius[9]	370	70	18.9
Cooksey[10]	53	21	39.6
Rosenbaum and Levine[11]	208	68	33
Woods and Barnes[2]	128	60	46.9
John Sealy Hospital[12] cases	85	40	47
Total	1983	513	25.8

This figure of a 26 per cent mortality is probably as nearly accurate as can be obtained. If it is in error the likelihood is that it is too high, at least for those cases coming under medical observation, because even when cases are selected with the utmost care there will be included some in which previous attacks have occurred, and in them the mortality is invariably higher than in initial attacks. On the other hand there are many patients with coronary occlusion who die suddenly. The great majority of these are not studied at necropsy, and hence are not correctly diagnosed. Some figures are available that may help in clarification of this unknown factor. In a recent report Kruse[13] found that in 1718 sudden, violent and accident deaths in which necropsies were performed, 869 were due to "natural causes." Of the latter 556 were cardiac deaths and of them 338, or 39 per cent (of those due to "natural causes"), were due to disease of the coronary arteries. The pathologic findings in the coronary artry disease cases were summarized as follows: coronary occlusion occurred in 181 cases, coronary sclerosis in 70, and coronary thrombosis in 60 cases.

Taking into consideration the probability that approximately 40 per cent of sudden deaths due to "natural causes" are the result of coronary occlusion, the mortality rate for the acute phase of the disease must be revised upward. It seems reasonable to assume that it lies somewhere between 30 and 35 per cent. Therefore, the person who suffers a coronary occlusion is confronted with the probability of better than a 2:1 chance of immediate survival.

What then, of the 65 to 70 per cent who outlive the acute attack? In his classic description, "Clinical Features of Sudden Obstruction of the Coronary Arteries," James B. Herrick,[14] in 1912, clearly recognized the possibilities with respect to recovery. He said: "Death is the result in nearly all cases. Yet it may be delayed for many days. More than this, there is, as has been shown by reference to experimental work, no intrinsic reason why some patients with obstruction of even large branches of the coronary artery may not recover. Experimental animals sometimes do. And as already said, mild cases must occur, and one cannot pretend to say where the dividing line should be drawn between the mild obstruction of a coronary branch, whose recovery means a few fibrous patches in the

myocardium, and the more serious one that in a few days is to lead to rupture of the heart or is to produce an extensive weakened fibrous area that will ultimately yield in cardiac aneurysm." Since Herrick's monumental contribution 30 years ago we have learned that fully two-thirds of the patients do survive the initial attack.

Furthermore, it is a matter of common information that many patients have a long survival. One of my cases recovered from a severe coronary occlusion 15 years ago, and is now leading a busy, useful life free from symptoms and signs of heart disease. Others are carrying on eight to ten years from the date of their occlusions. White[15] reported the case of a man who died in his seventy-third year, 25 years after his first coronary occlusion, and Drake[16] cites one who died suddenly at the age of 80 from a nocturnal attack of angina pectoris nearly 40 years after the initial attack of myocardial infarction.

A number of writers on this subject record instances of long survival up to 20 years or more, but such cases are admittedly the exception. Drake[16] concluded, from the published figures from "several large groups" that the average duration of life is slightly over two years. Levine and Rosenbaum[17] found 41.1 months to be the average survival time of 101 patients studied by them, and Palmer's[18] 65 patients who were followed till death lived an average 4.2 years. When it is recalled that the most frequently observed coronary occlusion patient is between 50 and 60 years of age at the time of the first attack, it will be readily appreciated that many will reach an age equal to or beyond a normal life expectancy.

It is a well established fact that the great majority of patients who have a coronary occlusion die from heart disease in some form. Apparently more than one-third succumb to a subsequent attack of myocardial infarction if there are accepted the established cases plus a somewhat indefinite number that die suddenly, since obviously some of the latter have termination due to unproved coronary occlusion.

ANGINA PECTORIS

Angina pectoris precedes coronary occlusion in from 22[9] to 72[11] per cent of patients reported in various surveys. It also occurs frequently after an occlusive episode in some instances in which it was previously absent. In neither event is there conclusive evidence to show that the post-occlusive average duration of life is shortened, though without doubt such patients experience more subjective symptoms, and are more seriously incapacitated than those without a post-coronary occlusion anginal syndrome. Patients with angina pectoris furnish a considerable number of cases of sudden death, some due to a massive myocardial infarction, some due to ventricular fibrillation and others as now seems well established to cardiac standstill.[19] A small percentage of persons who present a history of angina pectoris prior to the occlusion experience no anginal attacks after recovery. This is not well explained. In some it appears to be due to a lowering of the blood pressure, but in others no suitable explanation can be given. In any event such an occurrence has no prognostic significance.

CONGESTIVE HEART FAILURE

Another group of coronary occlusion cases die after an interval of months or years from congestive heart failure. In Palmer's[18] series it accounted for 13 per cent, in that of Levine and Rosenbaum[17] it caused death in 20 per cent, and in 370 cases studied by Willius[9] "gradual cardiac failure" was the mode of exitus in 52 per cent. Palmer[18] stated that in the group of 212 cases observed by him congestive failure occurred only in those with enlarged hearts, and furthermore that following recovery from an attack of coronary occlusion the probability is twice as great that the patient with a normal sized heart will be able to lead an active life as one with an enlarged heart. Hypertension existing either before or after myocardial infarction appears to exert but slight influence on the longevity except as it may be the etiologic agent in cardiac enlargement.

The cardiac death rate increases steadily with each succeeding attack of coronary oc-

clusion. As previously shown about one-third die from the first infarction; the rate doubles for second attacks, and approximately three-fourths fail to survive a third. Of Willius'[9] cases 80 per cent had a single coronary occlusion, in 17 per cent two episodes occurred, and 2.2 per cent had a third. Some series give higher percentages than those for multiple attacks. On the basis of these figures it may be asserted that patients surviving an initial attack of coronary occlusion have a greater likelihood of subsequently developing other cardiac complications, notably angina pectoris and congestive heart failure, rather than further attacks of myocardial infarction. As regards these two most frequently encountered end results, angina pectoris is much more common, but it does not appear to shorten greatly the period of survival, whereas in congestive failure following coronary occlusion the average survival period is reduced by about 25 per cent. It was pointed out by Levine and Rosenbaum[17] that congestive failure occurs more than twice as frequently with anterior infarctions as compared with posterior lesions.

A very small percentage of recovered coronary occlusion patients die non-cardiac deaths, about 5 per cent in most series. This emphasizes strongly the fact that once a myocardial infarct of appreciable extent has formed the threat of future disaster from heart disease in some form is an ever present possibility.

So far, consideration has been largely given to the unfavorable side of the picture. What is the future for those surviving an initial attack or even subsequent ones? Slightly more than half the patients will be able to return to work, either part or full time. Of this group about 50 per cent experience mild anginal seizures, dyspnea or weakness to a degree not incompatible with ordinary activity.

SUBSEQUENT CARE

The average patient should be kept at complete rest for from six weeks to three months after a coronary occlusion. Following this period another three to six months should be required to restore gradually the individual to former activity, and/or to determine his fitness for or limitations concerning future effort. As Master, Dack and Jaffe[21] have emphasized, and apparently proved by their investigations, effort, or other preceding states have nothing to do with initiating the attack of coronary occlusion which is an end result of atherosclerosis of the coronary arteries. Therefore, there is no reason to believe that factors involved in the occupational status are more than coincidental in favoring the onset of coronary occlusion, nor are these same factors likely to precipitate further attacks after recovery and the resumption of activity. It does go without saying, however, that hard labor is usually not so well tolerated after recovery from coronary occlusion because of the incident lowering of the cardiac reserve in many instances. On the contrary, experience has amply proved that it is decidedly advantageous to patients to resume some, if not all, of their former activities. The tendency of cardiac patients to develop a state of chronic and frequently unjustified disability and invalidism as much from psychogenic reasons as from the heart disease itself is too well known to require comment. The duty of the physician is to offset this as far as possible, consistent with sound principles. It is his obligation to rehabilitate the patient as much as it is to apply sane and judicious therapeutic practices in the course of the acute illness.

There is no justification for the belief that coronary occlusion alone constitutes total permanent disability, even though its complications and sequelae may do so. All too frequently patients who have made complete recoveries are confronted with the difficulty of returning to their former jobs, after myocardial infarction, because of the restrictions imposed by employers' liability under workmen's compensation laws. It has been suggested[22] that those so affected should, in fairness to themselves and their employers, sign waivers removing liability for subsequent cardiac illness. This would overcome some of the present obstacles to the restitution of many persons with coronary artery disease. By the same token

fully recovered patients should not be certified as totally and permanently disabled in order that they may take advantage of disability insurance, unless a gradual and conscientious effort to restore them to activity has failed.

In 1939 the American Heart Association appointed a Committee to Study Coronary Artery Disease. This committee compiled a large amount of information from a total of 228 replies to a comprehensive questionnaire that was sent to outstanding cardiologists and teachers of medicine. The overwhelming majority opinion was:

1. Every person who has sustained a coronary occlusion is not totally and permanently disabled.

2. From 50 to 80 per cent of patients who have recovered from coronary occlusion may be permitted to attempt partial or complete resumption of activity.

3. That angina pectoris and congestive heart failure are the most important handicaps to a resumption of activity.

4. Patients who have recovered from coronary occlusion, but who have angina of effort should be treated with especial precautions to prevent chronic invalidism.

5. Effort that causes no cardiac symptoms does not shorten the life of the patient who has recovered from coronary occlusion.

6. A return to business or profession, with some modification of activity, does not exert an unfavorable influence on the course of coronary disease, nor does it increase the tendency to further coronary occlusion or shorten life.

7. The fully recovered patient may be permitted to return to business or professional activities in from three to six months.

8. It is safe for patients who have recovered from coronary occlusion to drive automobiles.

9. That recovered coronary occlusion patients may be allowed to return to occupations covered by industrial law, with restrictions.

CONCLUSION

It may be said that the immediate fate of an individual stricken with coronary occlusion is difficult to predict. Certain easily obtainable evidence may be of assistance in arriving at a fairly reliable prognosis. At least two-thirds of the cases recover, and of them a majority may be restored to an active and useful life without undue hazard resulting therefrom. A careful study of the facts does not warrant a hopeless outlook for the patient who has survived the initial attack, but, on the other hand a degree of enlightened optimism is not unreasonable.

REFERENCES

1. Shillito, F. H., Chamberlain, F. L., and Levy, R. L.: Cardiac infarction: the incidence and correlation of the various signs with remarks on prognosis, J.A.M.A., 118: 779, 1942.

2. Woods, R. M., and Barnes, A. R.: Factors influencing immediate mortality rate following acute coronary occlusion, Proc. Staff Meet., Mayo Clin., 16:341, 1941.

3. Silberberg, M. D.: Prognosis in coronary occlusion, M. J. Australia, 1:298, 1938.

4. Conner, L. A., and Holt, E.: The subsequent course and prognosis in coronary thrombosis, Am. Heart J., 5:705, 1930.

5. Clark, L. J.: Prognosis in coronary disease, New Orleans M. & S. J., 86:365, 1933.

6. Levine, S. A.: Coronary thrombosis: its various clinical features, Medicine, 8:245, 1929.

7. Howard, T.: Coronary occlusion: based on the study of 165 cases, M. Times and Long Island M. J., 62:337, 1934.

8. Master, A. M., Jaffe, H. L., and Dack, S.: The treatment and immediate prognosis of coronary thrombosis (267 attacks), Am. Heart J., 12:549, 1936.

9. Willius, F. A.: Life expectancy in coronary thrombosis, J.A.M.A., 106:1890, 1936.

10. Cooksey, W. B.: Coronary thrombosis: follow-up studies, with especial reference to prognosis, J.A.M.A., 104:2063, 1935.

11. Rosenbaum, F. F., and Levine, S. A.: Prognostic value of various clinical and electrocardiographic features of acute myocardial infarction: immediate prognosis, Arch. Int. Med., 68:913, 1941.

12. Herrmann, G. R., and Deckerd, G.: Acute coronary occlusion with cardiac infarction: Some diagnostic and prognostic and therapeutic suggestions, Southern Med. J., 32:696, 1939.

13. Kruse, W. E.: A statistical analysis of 1700 sudden and violent deaths, J. Indiana M. A., 33:509, 1940.

14. Herrick, J. B.: Clinical features of sudden obstruction of the coronary arteries, J.A.M.A., 59:2015, 1912.

15. White, P. D.: A new record in longevity after coronary thrombosis, J.A.M.A., 108:1796, 1937.

16. Drake, E. H.: Long survival following coronary thrombosis, Am. Heart J., 20:634, 1940.

17. Levine, S. A., and Rosenbaum, F. F.: Prognostic value of various clinical and electrocardiographic features of acute myocardial infarction; ultimate prognosis, Arch. Int. Med., 68:1215, 1941.

18. Palmer, J. H.: The prognosis following recovery from coronary thrombosis, with special reference to the influence of hypertension and cardiac enlargement, Quart. J. Med., 6:49, 1937.

19. Goodrich, B. E., and Needles, R. J.: Terminal cardiac mechanism in coronary artery disease, Am. Heart J., 20:637, 1940.

20. Master, A. M., and Dack, S.: Rehabilitation following acute coronary artery occlusion, J.A.M.A., 115:828, 1940.

21. Master, A. M., Dack, S., and Jaffe, H. L.: Activities associated with the onset of acute coronary artery occlusion, Am. Heart J., 18:434, 1939.

22. Ungerleider, H. E.: Discussion of paper by Master and Dack, reference 20, J.A.M.A., 115:832, 1940.

DISCUSSION

Dr. A. A. Herold (Shreveport): Dr. Stone has brought us a very timely subject, one which should be of interest to any medical gathering. He has talked about a doctors' disease. As you know, I am sure, a large per cent of doctors die of heart disease, most of which is coronary trouble. I hope you will pardon a personal illustration, referring to first person singular. I would say this subject was very interesting to me last summer. I went to bed with what some of my confrères called a coronary attack. "The future of the coronary thrombosis patient" was uppermost in my mind!

Dr. Stone gave some very interesting guides in prognosis. Let me say here that I think the hardest thing we have to deal with in the practice of medicine is prognosis in general. I know you have experienced this in these heart cases. He stressed high temperature, high leukocytosis and venous pressure as important guides. Low pulse pressure and rising venous pressure are important.

Cardiographic studies very often are misleading in prognosis, especially if taken very soon after the supposed attack. I have seen doctors misled by taking an electrocardiogram 48 hours after an attack in which the definiteness is not there and they have to be studied further. That is where the cardiogram comes in, for comparative purposes.

Dr. Stone also told you that this condition is more common in men than in women. I am sure that is our experience too.

Another form of coronary occlusion to which he has referred, not due to thrombosis, is gradual closing with sclerosis. I had such a case, a man with recurring attacks, diagnosed coronary thrombosis. At autopsy, there was no evidence of thrombosis; coronary arteries firmly closed and when we succeeded in opening with scissors, the sclerosis was so complete, it grated on the instrument.

THE RATIONAL MANAGEMENT OF BRONCHIECTASIS*

CHARLES R. GOWEN, M. D.
SHREVEPORT

In discussing the management of bronchiectasis, it is well to mention briefly the etiology, the prophylaxis, the pathology, the symptoms, the diagnosis and the prognosis. By understanding these, one is able to deal with this condition intelligently as it cannot be treated or managed without a knowledge of the causative agents and the mechanical factors involved in this condition. All of these will be briefly reviewed at this time.

*Read before the sixty-third annual meeting of the Louisiana State Medical Society, New Orleans, April 29, 1942.

ETIOLOGY

The etiologic factors can be divided into three main classifications: (1) Defects in the bronchial wall from lack of development in early life; (2) inflammatory conditions; (3) mechanical strain and stress on the bronchial wall itself.

The discussion of embryologic changes that occur in the bronchial walls will be limited to the fact that any condition that prevents the complete aeration of the lung immediately after birth, or some unknown causes before birth, will have a tendency to weaken the bronchial wall and cause defects in varying degrees.

The condition that concerns the physician most is the inflammatory reactions in the lung at any period in life. From my own personal experience, I am sure that the handling of acute, subacute and chronic bronchial irritations of any kind at the early stage can prevent the development of a definite bronchiectasis or a bronchiolectasis in a large per cent of the cases later on. The relationship between the sinuses and the bronchial tree is definitely established by many workers and sinusitis can be considered one of the causes of bronchiectasis. The percentage varies with different workers. Many manifestations of allergy are definitely responsible for some cases of bronchiectasis.

In the third classification, that of mechanical strain and stress, can be placed atelectasis. In this instance, there is a definite negative pressure in the thoracic cage and a pulling apart of the bronchial walls, which causes dilatation. In the obstructive type would be neoplasm, foreign bodies and scar tissue from previous inflammatory changes in the bronchial walls, of which

tuberculosis and syphilis are the most frequent causes.

PROPHYLAXIS

In the first classification of bronchiectasis, there is very little we can do. Usually the damage is done before the condition is recognized. On the other hand, in the inflammatory type, such as any of the acute diseases that affect the bronchial tubes—influenza, bronchopneumonia, bronchitis and the acute respiratory infections—everything should be done to keep the secretions from accumulating and becoming organized so that they fail to drain all portions of the inflamed bronchial tree. The use of expectorants rather than sedatives, the liberal use of mild inhalations of steam plus postural drainage usually prevents this complication. If the secretions can be kept moving and the organization of any given portion of the bronchial tree prevented, it is impossible to affect the bronchial wall. We know the greatest damage comes from the infections of the lymphatics in and around the bronchial tubes, and the so-called interstitial pulmonitis plus the weight of secretions is considered the usual cause of the breaking down and weakening of the bronchial wall. The prevention of bronchiectasis in the obstructive type is perfectly clear, by the use of the bronchoscope. If possible, the clearing away of granulated tissues and new growths, the dilatation of the stenosis and sometimes a simple shrinking of the inflamed mucosa may relieve this condition. All cases of chronic bronchial conditions should have a careful check of the sinuses, teeth and all possible foci of infection and should be cleared up if possible.

PATHOLOGY

Considering the pathogenesis of bronchiectasis, one must realize that there are varying tissue changes in the bronchial tubes according to the type of dilatation. It has been demonstrated repeatedly that at times there is a definite diverticular type of dilatation where there is a break in the bronchial wall or an actual tear into the lung tissue. In other areas, there will be a definite dilatation or stretching of the entire circumference of the bronchial tubes. In other instances, the interlining remains intact but there is a giving away of the deeper structures and the lowering of tone in the bronchial muscle wall and its elastic tissue. At another point, there is the breaking apart of the bronchial wall which forms a rather definite cavity and gives the appearance, when filled with iodized oil, of a sacculation. The age of these lesions determines to a great extent the type of cell lining them.

In studying a large number of slides prepared by Dr. Matthews of the Shreveport Charity Hospital, we have found these various types existing in the same lung. All granulated tissues replacing normal bronchial structures are highly vascular and new vessels directed toward the surface have fibers of scar tissues running throughout like a skein. The parenchyma of the lung in this area adjacent to the dilatation is usually slightly collapsed. There is also a marked increase in the secretory glandular structures which accounts for the profuse expectoration. The cartilage itself is not grossly affected but at times markedly displaced. Patches of emphysema are also noted. Endarteritis and lymphoid hyperplasia are conspicuous.

SYMPTOMS AND SIGNS

The classical symptom is a severe cough with profuse expectoration which separates out into three layers; a frothy layer at the top, liquid for the second layer and a sediment at the bottom layer. From time immemorial this has characterized the classical description of a bronchiectatic sputum. This only occurs in the long-standing, well-established case with good drainage and certainly fits that picture most admirably.

The case that gives the physician most concern is the border line patient who has repeatedly attacks of respiratory infection with visible increase in cough, increase in sputum and at times obviously sick for several days until the sputum loosens up and begins to drain out. In the bronchial tree, as in any other condition, free drainage of

all infected areas is absolutely essential. Due to new blood vessels forming in granulation tissues and the breaking of blood vessels of the bronchial wall, the sputum is often blood-streaked. Occasionally there is a free hemorrhage if a large blood vessel is injured.

The physical signs of bronchiectasis will not be discussed here as they vary so much that it is impossible to discuss them intelligently. The symptoms can be classified and will fit any acute or chronic lung condition and must be differentiated from tuberculosis, syphilis or bronchial carcinoma, which may complicate it at any time.

DIAGNOSIS

The diagnosis of bronchiectasis can only be established by filling the bronchial tree with an opaque substance and studying it roentgenographically, giving a clear picture of just what is being dealt with. Any other method falls short. The method used is rather simple and may be varied according to the doctor's own choice—any way that he can get an opaque substance into the bronchial tree is sufficient. Before a rational conclusion can be reached, all lobes of both lungs should be visualized.

Iodized oil should be introduced into the bronchial tree and the patient should be placed in different positions to visualize all lobes of both lungs. I have found that it is always a safe procedure to test an individual for iodine sensitivity before injecting the iodized oil into the bronchial tree. It is also best to give an expectorant and have the patient drain all possible areas in order to allow the oil to penetrate out even to the smaller bronchioles. If this is not done, the complete picture cannot be obtained. The bacterial flora found in the expectoration changes from time to time and does not give one a clear picture of the actual cause. It has been found in a large percentage of cases that the Vincent's spirochetes are in predominance in the sputum; whether they are primary or secondary is open to question. The various forms of yeast and fungi are also present, and as mentioned before, change from time to time. The amount and character of sputum raised is some guide as to the size of the dilatation but might be of very little diagnostic value since there is an increase in the glandular structures in the dilated bronchial tubes and the only possible way to be sure that one is dealing with a dilatation of the bronchi or bronchiole is to study the exact size of the tube after it has been injected with iodized oil.

PROGNOSIS

The prognosis of the case depends on the extent and location of the diseased bronchi. Once a bronchus or bronchiole is dilated, there is no way of bringing this tube back to a normal bronchial tube. In recent years, thoracic surgery has promised relief now to a great many people who otherwise could not have regained anything but a fair degree of health. A lobectomy or pneumonectomy does not carry the same mortality now that they did several years ago and they should be considered in every case in which there is a fair chance for success. Pneumothorax also offers relief and in some cases, an apparent relief of symptoms, but it has not been used in a large enough number of cases in my experience to warrant recommending it. Phrenic interruptions and phrenicectomy also have promised marked benefit to a great many cases. The main object of this discussion is the management of a case which will not and does not warrant more radical surgical procedures. We all know of a large number of individuals with dilated bronchi who go through life and live out their expectancy, but their comfort and longevity will depend on how well they keep the bronchial tree drained.

TREATMENT

The real management of bronchiectasis is definitely divided into drainage, specific medication, supportive treatment and radical operative procedure. X-ray is of questionable value.

Postural drainage heads the list always and should be carried out every three or four hours in an acute exacerbation and then as often as necessary in the individual case to keep the patient comfortable and

relieve the annoying hacking cough. To accomplish the proper drainage, the patient should be in the following position: The mouth should be at least 18 to 20 inches below the base of the lung and the patient should be instructed to turn from side to side. He should breathe deeply and make every effort to cough. This will force air distal to the secretions and cause it to be expelled. This position should be maintained until the bronchial tree is thoroughly emptied. At times, where there is an obstruction, a constriction or narrowing, a bronchoscope should be passed and whatever obstruction is found should be cleared out, especially if this is a new growth or some foreign body. Often patients are relieved by dilating the bronchial tubes with adrenalin and pontocaine with the aid of mechanical dilators. The secretions can be aspirated also. There has been considerable work done with the use of iodized oil in the actual treatment of bronchiectasis with satisfactory results. The iodized oil displaces the sputum and, being heavier, dislodges it and it is expelled with a lessening of the toxic symptoms. This is only temporary and should not be repeated too often as there is a possibility of getting some iodine reaction.

Specific medication might be classified as any medication that would change in a limited degree to the character of sputum. Some of the milder creosote preparations have been helpful, providing they are tolerated by the patient's gastrointestinal tract. Ammonium chloride is the most useful preparation as potassium iodide might weaken the already inflamed bronchial wall. Autogenetic vaccines have given considerable relief, if the material is obtained through a bronchoscope. An occasional use of neoarsphenamine intravenously or bismuth intramuscularly, when there is a definite Vincent's spirochete found in the sputum, could be recommended.*

*Sulfonamides are of use if the predominating organisms are streptococcic and pneumococcic but only in the acute exacerbations.

In supportive treatment and general care, the patient should be told to take definite rest periods, avoid strenuous exertion to the point of marked dyspnea, avoid exposure and dust. If the patient suffers from any upper respiratory disturbance, such as chronic sinus or pyorrhea pockets or chronic tonsillitis, he should take measures to have them corrected since it has been proved there is a direct connection through three open channels—lymphatic, bronchial and blood stream. The bronchial tree is affected by any inflammatory condition in the sinuses opening into the upper air passages, the teeth and the tonsils and we cannot expect to get relief or results without getting at the cause of the condition.

SUMMARY

The surgical procedures that have proved of value are pneumothorax, phrenic interruptions, lobectomy and pneumonectomy, but these do not come in the scope of this discussion. It might be well to mention that the age of the patient, his general condition and the various circumstances surrounding the patient would make the decision on the course or type of operation indicated.

DISCUSSION

Dr. Julius L. Wilson (New Orleans): I enjoyed Dr. Gowen's paper very much because it was a practical paper. He has based it upon practical experience. Most of the papers on bronchiectasis get into too much theory and not enough of the actual management of cases. In childhood and youth the only curative treatment is surgery. The management of these cases, 60, 50, and 40 years old is another and very difficult matter. I think he rightly emphasized postural drainage as one measure for clearing the bronchial secretions. Postural drainage should be the first treatment of these cases that have chronic expectoration,—not only postural drainage for three or four hours, but constant postural drainage, with the foot of the bed elevated 18 inches on shock blocks. Giving constant drainage is very valuable over a period of a week or two. Bronchoscopy is a very important supplement. Personally, I am opposed to the routine use of lipiodol. It is not curative; it is palliative. Another method of producing expectoration, which can be applied more safely, is the use of ammonium chloride or potassium iodide by mouth. I believe that we may do harm by the introduction of lipiodol, a foreign body, into the lung. Certainly, it is bad medicine when it spills

over into the more normal bronchial tree and gets into the alveolar structures of the lung.

The sulfonamides are helpful and should be used when the patients have a pneumonitis. In some cases preoperatively they will help clear infection. We have not been impressed with the number of cases that have spirochetes and fusiforms. In most cases of long standing bronchiectasis the search for spirochetes in the sputum is not successful. Mechanical obstruction of the bronchus favors infection, and anaerobic infection may be an important factor. In such cases, the use of arsenicals may be indicated.

I think this paper is most valuable, particularly in the management of older patients not suitable for operation, and in the treatment of the younger patient before operation.

One word on pneumothorax. Some years ago, Dr. Rist advocated pneumothorax, particularly in children, and there was a wave of enthusiasm for this treatment. Pneumothorax can seldom be completed because of adhesions. If it be successful, we get a benefit which lasts as long as we maintain the pneumothorax and then find that there remain the same pockets which existed before. So pneumothorax is generally disappointing in these cases.

Dr. J. Preston Davis (Lake Providence): The speakers mentioned the use of arsenicals in certain cases of bronchiectasis. I know that bronchiectasis and bronchial asthma are separate entities, but it is my impression it is very dangerous to use in bronchial asthma. I would like to know about this.

Dr. Randolph Lyons (New Orleans): I would like to ask Dr. Gowen and Dr. Wilson if they could give me some idea, approximately, of the mortality in lobectomy in the surgical treatment of bronchiectasis. Dr. Wilson suggested that it be done in young patients because the operative risk would be a great deal less. I feel that even in middle aged persons there is a great deal of risk.

A patient I saw in consultation was operated on for bronchiectasis. She was a woman whose main symptoms were discomfort, chronic cough, and expectoration. She was a housewife and able to look after her affairs. She decided to have the operation done and died several days after the lobectomy of pneumonia of the opposite lung. I was asked to see the patient because of the involvement of the remaining lung. This was an unfortunate result but should not discourage operation in severe cases.

I would like to have an idea of the mortality of the operation in good hands.

Dr. Charles R. Gowen (in closing): In regard to the use of arsenicals in any bronchial condition, as I explained in my first discussion, I was only trying to discuss the management of a definite bronchiectasis so diagnosed. Now, in the various forms of allergic bronchitis and chronic bronchitis, I do not think at any time we should inject arsenicals into the vein which we know goes directly into the heart, and the next second highest concentration of that product is around the bronchial tree throughout the lung, and we get the maximum reaction there. If the patient is slightly sensitive to arsenicals we will get increased reaction. We use arsenicals in chronic bronchiectasis where there are spirochetes, but when we get into asthmatics we are dealing with a different situation.

As to Dr. Lyons' question regarding operative mortality, I can remember and most of you can too, when surgeons first started doing lobectomy. The mortality has dropped consistently since then. I think about the best figures are 4 to 5 per cent.

The preparation of the patient and the postoperative care, more than the technic of operation, have more to do than anything else. These cases can be managed, and if the individual is going along fairly comfortabiy, do not operate, because the closing of the cavity in the lung in lobectomy is not keeping the patient on his feet.

—o—

WAR WOUNDS OF THE CHEST*

HARLON W. HARRISON, M. D.
SHREVEPORT

Modern warfare, with the indiscriminate bombing of the civilian population has made every physician a potential military surgeon. In past wars, wounds of the chest have caused a very high percentage of the deaths and there is every reason to believe that with aerial bombs and high explosive shells playing an increasingly important part in warfare, that chest wounds will become even more important. Hoche has shown that while among the eleven million American, English, French and German soldiers wounded in World War 1, only 6 per cent suffered from wounds of the thorax; the mortality from such wounds was 56 per cent. This was second only to wounds of the abdomen which was 68 per cent. The mortality from all wounds was 8 per cent. Trueta has shown that in the recent civil war in Spain among 9,850 patients treated in the hospitals of Barcelona, 12.1 per cent

*Read before the sixty-third annual meeting of the Louisiana State Medical Society, New Orleans, April 30, 1942.

had thoracic injuries. Crafoord states that wounds of the chest are still responsible for from 30 to 40 per cent of all war fatalities.

Ranson has presented the interesting fact that as the result of improvements in the method of handling war wounds of the chest, the mortality has steadily decreased. In the Crimean War the English lost 79.2 per cent of the soldiers who suffered from wounds of the chest. In the American Civil War the mortality was 62.6 per cent; the Germans in the Franco-Prussian War 56.7 per cent; British in the last war 27.5 per cent, and the Chinese in the Sino-Japanese War 14.8 per cent.

It is very probable that this improvement has resulted to a large extent from the increased knowledge of the physiology of respiration as well as the improvement in surgical technic generally. No better example can be found of the fine results which can be obtained by the proper and prompt handling of war wounds than that reported by Moorhead in his "Experiences at Pearl Harbor."

In order that we may better understand the reason why certain measures are necessary when we are confronted with war wounds of the chest, we must understand something of the physiology of the chest, as it influences surgical procedures, and we must also understand something of the forces and missiles which produce war wounds.

PHYSIOPATHOLOGIC CONSIDERATIONS

The thorax is a relatively firm bony cage containing vital organs whose efficient function is dependent upon the maintenance of a normal negative intrathoracic pressure. Wounds of the chest, and especially war wounds, as they are usually more severe and more extensive than those in civil practice, often upset this balance. Thomas has outlined the conditions producing this interference as follows: (1) The presence of air or blood in the pleural cavity under sufficient pressure to encroach not only on the homolateral but also the contralateral lung as a result of the ensuing mediastinal displacement, this displacement at the same time producing severe cardiac disability, chiefly by inefficient filling as a result of distortion of the superior and inferior vena cava and the absence of the aspiratory effect that the negative intrathoracic pressures usually exerted on these structures. (2) Open pneumothorax: The ill effects of this condition briefly stated are: (a) Lung collapse of the affected side; (b) pendulum swing of air during respiration from the expanded to the collapsed lung and vice versa; (c) mediastinal flutter and paradoxical swing of the mediastinum during inspiration and expiration with the stimulation of the contained cardiac and pulmonary plexus; (d) loss of aspiration on the great vessels leading to deficient diastolic filling of the heart, in its turn leading to deficient cardiac output. (3) Cardiac tamponade: This results from fluid in the pericardial sac constricting the heart and preventing efficient diastolic filling. (4) Stove in chest, producing essentially the same effect as an open pneumothorax by allowing for paradoxical respiration.

Just as wounds of the chest differ from wounds of the other parts of the body, so do war wounds of the chest differ from wounds of the chest seen in civil practice. The former are usually more severe and more extensive. We can hardly treat war wounds intelligently without at least an elementary knowledge of the agents which cause them. Projectiles can be divided into three main groups: (1) rifle, revolver and machine gun bullets; (2) shells from artillery, and (3) bombs. To this list of common methods of war trauma we must add crushing by falling masonry and the pressure by "blast." Both of these latter are largely the result of the development of aerial warfare.

ACTION OF SEVERAL TYPES OF MISSILES

Bullets from rifles and machine guns are all essentially the same. The range of the modern rifle is from one and seven-eighths to two and a quarter miles. The muzzle velocity of the German rifle bullet is 2800 feet per second, but after traveling for about 600 yards this velocity is reduced to 1100 feet per second. The velocity of re-

volver and submachine gun bullets is considerably less. If a bullet drills a clean hole it gives the surgeon relatively little trouble unless a vital organ or a large vessel is struck. As the velocity of a bullet decreases, however, it begins to wobble and may even turn end over end due to air resistance. If such a bullet strikes in such a position as to have the long axis of the missile at right angles to its direction of flight, the damage may be more extensive. Usually the wound of exit, if there is one, is much larger than the wound of entrance.

Projectiles fired by artillery are high explosive shells. These consist of thick iron casings enclosing a quantity of violently explosive trinitro-toluene. Bursting is brought about by means of a detonator which comes into action by impact. The fragments vary in size from minute particles to pieces weighing several pounds. The shells weigh from 15 to 120 pounds and break into 600 to 1500 fragments. These pieces are jagged and when they penetrate the chest wall they produce large wounds with extensive tissue damage. They often carry fragments of clothing into the wound ahead of them.

Aerial bombs are to a great extent a product of the present conflict and in this respect this war differs from its predecessors. This brings several new factors into the picture. First, as the result of the important part played by the aeroplane, the fighting man (and the civilian) is as much exposed to the enemy above him as he is to the enemy in front of him. The aerial bomb breaks into innumerable tiny fragments and is as apt to strike in back of a person as in front of him, and the person may suffer from multiple wounds on either the anterior or posterior surface of his body. Someone has said that this conflict is "the war of the crouching man" because when a person hears enemy planes overhead he instinctively crouches or falls to the ground.

There are three other effects resulting from aerial warfare. One is, since the aerial bomb is not subjected to the same great force that the shell is when it is fired, it does not need the heavy casing that the high explosive shell does. As the result the relative weight of explosive contained in the aerial bomb is much greater. When the explosion of one of these bombs occurs the fragments are much smaller and have a much higher velocity than do other types of projectiles (4,000 feet per second). Even though these particles are small, it has been found that even small pieces traveling at such a velocity have a devastating effect on tissue. While on the surface there may be only a trifling wound, beneath the skin there is an extremely widespread destruction. The effect is similar to that produced by an internal explosion.

A second effect of the aerial bomb is the fact that it causes buildings to collapse (a ton bomb leveling almost everything in a city block) and the falling masonry causes many crushing injuries.

A third effect of the violent explosive aerial bomb is "blast." It results from the detonation of the bomb. Zucherman states that in such an explosion the solid explosive is converted into gases which are confined in the casing at a pressure which has been estimated as between 100 and 650 tons per square inch. As a result of this pressure the casing is blown to pieces, the gases escape, and by their expansion produce a blast wave in the surrounding air. The blast has two components, the pressure exponent and the suction component. This force causes extensive damage to the lungs. This will be considered later under the treatment of the various types of wounds.

Bayonet wounds are comparably infrequent. During the 1914-1918 war, wounds from bayonets comprised less than 5 per cent of the wounds. In this conflict the percentage will probably be lower. In fact there is a motion on foot to discard the bayonet. The Germans have done this already, supplying in their place, daggers.

TREATMENT OF THE COMMON WOUNDS

It is impossible within the scope of this paper to cover completely all the literature on wounds of the chest or consider in detail all of the various forms of injuries which may occur. An excellent exhaustive review of the literature has recently been presented

by DeBakey. I will attempt to mention only the more common conditions which may exist and outline their treatment.

Wounds of the chest may be divided into non-penetrating and penetrating wounds. In this conflict we are seeing more of the former than in previous wars. This is due to falling masonry and blast, and the damage caused to the intrathoracic viscera may be extensive without any apparant damage to the chest wall. These lesions are commonly associated with rather severe shock, and sometimes wtih serious hemorrhage which is not immediately perceptible.

Berry has stated that the principles underlying the treatment of thoracic injuries are: (1) The treatment of the surgical shock and hemorrhage, if present, by institution of appropriate measures and (2) the restoration of the thoracic viscera and the cardio-respiratory physiology to their normal status as soon as possible. Blades has emphasized that shock in severe chest injuries differs somewhat in manifestations and management from that observed in wounds of other parts of the body. In the former the mental condition is usually active and anxious and because of possible mechanical interference with vital capacity due to hemorrhage or to an open pneumothorax, dyspnea or even cyanosis may be prominent manifestations. Painful respiratory movements and coughing may further increase these difficulties. For these reasons first aid measures must include not only the treatment of the primary shock, but also the correction of the factors causing respiratory difficulty.

The pain and restlessness resulting from these wounds are greatly relieved by the use of morphine. It has been found that it is usually advisable to give 1-2 grain as the initial dose. The patient should be placed in a comfortable position, which is usually the sitting or propped up position in chest injuries, because breathing is easier in that position. The chest should be immobilized. This can be accomplished by multiple adhesive straps, multiple tape binder, or as Blades suggests, the complete encircling of the chest with a wide strip of adhesive at the level of the costal margin. This tape is applied on expiration. Prompt and adequate intravenous fluids, preferably plasma or blood should be administered to combat shock. The patient should be kept warm. During the period of shock, especially before the tape is applied, the patient should be rolled toward the affected side so that respiratory motion is limited on that side and any paradoxical motion is reduced to a minimum. The administration of oxygen is a valuable adjunct in the treatment of shock and relief of dyspnea and cyanosis. Transportation is very hard on patients suffering from chest wounds and Roberts and Tubbs state that in England it has become the established practice to move only those patients who need early primary operation. Even air transportation is bad for chest cases, where there is a pneumothorax present.

Hemorrhage, if present, must be controlled by ligation or in some other way stopping the flow of blood, and replacing the blood by transfusion with whole blood or plasma.

Wounds of the non-penetrating type are becoming increasingly more important due to falling masonry and the effect of blast. When a person suffers from a non-penetrating, especially a crushing injury, of the chest extensive damage can be done to the viscera with no perceptible injury to the chest wall. This is especially true with children whose chest walls are soft and elastic. The heart, lungs or great vessels can be torn with no damage to the chest wall.

Probably the most simple thing that can occur is a simple fracture of a rib. The easiest way to treat these injuries is by strapping the chest with adhesive. This can be done by placing one wide band all the way around the chest at the level of the costal margin or by placing several overlapping strips over the site of injury and extending three or four inches beyond the midline both anteriorly and posteriorly. If a rib fragment is displaced it can be brought back in place by putting a towel clip around it and exerting traction. This is rarely necessary. Latteri has suggested

injecting the intercostal nerves with alcohol, thus placing that portion of the chest at rest. Harmon, Baker and Kornegay have suggested injecting procaine between the rib fragments to reduce pain, especially during transportation. If the rib is jagged it may tear an intercostal vessel. The treatment of this condition will be considered later as will the result of penetrating the lung with the rib end. When several ribs are fractured we have a "stove in" chest, and as this produces paradoxical respiration, it must be immobilized.

Fractures of the sternum are usually associated with injuries of the viscera and these usually must be taken care of first. They are usually also associated with considerable shock. The sternal fracture itself can usually be reduced by hyperextending the chest and neck with a sand bag between the shoulders. At the same time pressure is exerted on the protruding end of the overriding segment.

Bleeding from intercostal or internal mammary vessels which may be torn in nonpenetrating or penetrating wounds of the chest may be serious. It is commonly necessary to operate on these patients in order to find the bleeding point and tie it off. Intercostal bleeding can also be controlled by passing subpleural sutures around the rib both proximal and distal to this bleeding point. In an emergency, towel clamps may be clipped around the rib in a similar manner as a temporary "tourniquet."

Fractured rib ends may tear into the lung. This may cause hemorrhages into the pleural cavity, pneumothorax, subcutaneous or mediastinal emphysema. Subcutaneous emphysema is rarely serious, but it becomes too troublesome or if the tension on the skin is too great, multiple small incisions may be made through the skin to allow for the escape of air. If the leaking from the lung continues it may be necessary to do a thoracotomy and put a purse string around the tear in the lung.

When there is bleeding into the pleural cavity either from the lung or vessels in the chest wall or when air leaks into the pleural cavity from the lung, severe pressure symptoms may develop from the collection of blood or air present. As either of these increase in amount the homolateral lung is collapsed and the vital capacity is decreased. As more develops there may be a mediastinal displacement with displacement of the heart and collapse of the contralateral lung. Unless this pressure is relieved death may occur in a relatively short time as the result of the depletion of the cardiorespiratory reserve.

A tension pneumothorax may be relieved by repeated needle aspirations if the rate of leakage is not too rapid. If it is too rapid to be controlled by this means an intercostal catheter should be inserted and connected to a tube, the end of which is put under water to form a water seal.

There has been considerable disagreement concerning the best method to manage a hemothorax. Edwards and Davis state that 70 per cent of all chest wounds are thus complicated, so it is an important factor. In the past there has been a great tendency to leave the blood in the pleural cavity and allow it to absorb. This may be the best method in civil practice, where the damage is usually not so extensive, but even there the value of his regimen seems to be open to question. Weeks, of the New York police force, reports 76 cases of gunshot wounds of the chest which were treated without or with late aspiration with a 26 per cent mortality. In 17 persons who were treated with aspiration of the blood and replacement with air, there was no mortality. The disadvantages of leaving blood in the pleural cavity are: (1) An excellent bacterial medium is left in the pleural cavity, encouraging infection, and if infection does occur the empyema is very large; (2) the absorption of blood is very slow and the deposit of fibrin on the pleural surface often prevents reexpansion of the lung.

In England during the present conflict it has become the general routine to aspirate as much blood as possible about 24 hours after the injury (always before the fourth day). Fluid is removed as long as it can be obtained through a large bore needle. If

force is required, or if the patient complains of a pulling or tight sensation in his chest after a quantity of fluid has been removed, replace with some air. The use of a three way stopcock makes this easier and prevents the sucking of air into the chest as usually occurs when only a needle and syringe are used. Some authorities feel that air should always be replaced to prevent hemorrhage, but Roberts and Tubbs have found that they have no more recurrences of hemorrhage when air is not replaced, and in those cases where infection does occur, the empyema is much less extensive if air has not been replaced.

Blast injuries, as mentioned earlier, are due to the effect of the high pressure wave which is caused by the explosion of a bomb followed by a suction component. Patients suffering from blast commonly show no external sign of injury other than bloody fluid exuding from the nose and mouth. The patients are severely shocked, dyspneic, cyanotic, and usually complain of chest and abdominal pain. They are usually restless even after given 1-2 grain of morphine. Cough and hemoptysis are common. There is a limitation of motion of the chest and it seems to be held about three-fourths expanded. Attempts at deep inspiration are painful. Breath sounds are usually weak and many rales are heard. A few of these cases clear up, but in many intrapulmonary hemorrhage continues or pneumonia develops and death occurs.

Treatment consists of heat, morphine, oxygen and plasma transfusion. Operation is contraindicated unless absolutely necessary to correct some other condition. The patient should not be moved more than is imperative. Artificial respiration is absolutely contraindicated.

Traumatic asphyxia occurs as the result of direct crushing of the chest. It is characterized by deep bluish discloration of the face, neck and upper chest. There is no specific therapy and most patients recover completely.

In penetrating wounds of the chest there are several indications for primary operation. They are: control of progressive hemorrhage, convert an open pneumothorax into a closed one and prevent sepsis. Most thoraco-abdominal wounds require exploration.

Probably the most urgent indication is the patient with an open pneumothorax or a "sucking wound." If the opening is small, air may enter the pleural cavity on inspiration but not get out on expiration and as a result a dangerous tension pneumothorax develops. If the wound is larger there is a free ingress and egress of air, the patient will be in a critical condition due to collapse of the lung, mediastinal swing, or paradoxical respiration. Unless the opening is closed promptly the patient will die. In an emergency, a thick pad, preferably covered with vaseline to make it air tight, should be strapped over or sewed into the wound to stop it up. If there is hemorrhage the vessels can be tied off, or both the closing of the wound and the plugging of the wound and a stopping of the bleeding may be brought about by putting a mushroom shaped dressing into the pleura and then close the wound by an outward pull. If no further exploration is thought necessary the necrotic tissue at the wound edge is debrided away and the wound is closed. If an air-tight closure can be made without closing the skin, it is probably wise to do so, placing sulfanilamide in the wound and closing the wound by delayed primary suture at a later time.

If there is severe bleeding from the lung there is of course no question as to whether thoracotomy is indicated. However, for those inexperienced in the treatment of penetrating wounds it is difficult to decide which patients to operate on when the prevention of sepsis is the sole indication. Through and through wounds, when bony fragments or clothing have not been carried into the pleural cavity or lung, are unlikely to be followed by sepsis though Monod feels that exploration should be done more often than it is. While small fragments of shell may enter the chest in an intercostal space and cause little sepsis, it is probable that the dictum developed in the last war, "leave bullets alone, remove shell fragments," is a good one to follow.

If a thoracotomy is decided upon it may be done by enlarging the original wound, if it is in a position convenient for the exploration, or if not convenient a new opening may be made in an intercostal space or through the rib bed after the removal of several inches of a rib. The wound of entrance and the wound of exit, (if there is one) must be thoroughly debrided, or if early (a wound excision done) and any bone fragments removed.

After the thoracotomy opening has been made the ribs must be spread with a rib spreader. If the lung is wounded the various lobes are picked up separately and examined carefully. When the wounded portion is found it is brought out through the thoracotomy wound and the opening is packed shut around the exteriorized portion of the lung to prevent mediastinal swing. The tract of the missile is cleaned up and necrotic tissue trimmed away. Shell, rib and clothing fragments are removed. Bleeders or whistling bronchi are tied off. The pleura is sutured to prevent the escape of more air or fluid into the pleural cavity. If the damage to the lung is too extensive a lobectomy should be done, as the extensively damaged lung will probably undergo septic degeneration unless this is done.

If during the thoracotomy a rent is found in the diaphragm, this wound should be opened in a radial direction and the organs in the upper abdomen examined. If extensive damage is found, it may be necessary to do a laparotomy too. The diaphragm is then sutured.

Two new procedures are now being quite generally used and are proving to be very beneficial. One is poudrage of the pleural cavity with 10 to 15 grams of sulfanilamide before closing the wound. This is a convenient way of obtaining an early concentration of the drug as it is absorbed rapidly in the pleural cavity. A second is the introduction of a self retaining intercostal catheter to allow for the escape of air and effusion and obviates the necessity of postoperative pleural aspiration. This should be placed in the lower part of the chest, though not in the costophrenic angle as the diaphragm may obstruct drainage. Roberts and Tubbs state that the tube should not be put through the thoracotomy wound or the wound produced by the missile, but through a separate intercostal opening. Moorhead, however, stated that in the cases at Pearl Harbor the wounds of penetration were sewed tightly around the tube. In a letter from Dr. Moorhead he stated that "our chest wounds did well, and our wounds of entrance around the tubes showed no additional evidence of infection by comparison with those elsewhere." The catheter is removed at the end of 48 hours and the wound closed with a single suture, which had been inserted at the time of operation. If infection of the pleural cavity does occur it is treated as any acute empyema in civil practice.

WOUNDS OF THE HEART AND PERICARDIUM

If there is a large wound of the heart the patient usually does not live to reach the doctor. If the wound is less extensive, however, it is compatible with life if the condition is recognized and handled properly. The patient with a heart wound is commonly shocked but he may walk into the hospital under his own power. Patients with heart wounds usually show evidence of a wound in the vicinity of the heart. Prompt diagnosis is essential. The veins of the neck are usually distended. This is due to the accumulation of blood in the pericardial cavity whcih prevents the diastolic filling of the heart. There is a falling arterial and rising venous pressure. Heart sounds are distant or absent. Fluoroscopy shows diminished or absent activity. Treatment should be aspiration or operation. If the pericardial aspiration gives some relief which is followed by a recurrence of symptoms, or if it gives no relief, operation should be done at once. An incision is made over the heart. Usually it is necessary to ligate the internal mammary vessels. The pericardium is found to be distended and blue. The pericardial sac is opened, the blood and clots removed, and when the wound is found, this is sutured. Care must be taken not to include a coronary vessel in the suture. The pericardial sac is closed

as is the chest wall. These patients usually require transfusion during and after the operation. A patient with a wound of the heart should be given the chance to live by being operated upon, even if when seen by the surgeon pulse and respiration have apparently stopped. I have seen such patients start breathing and the heart start with massage after the pericardial sac has been opened.

Wounds of the mediastinum are difficult to handle. If large vessels are severed death usually occurs before the patient reaches the doctor. Foreign bodies in the mediastinum should be left alone unless they are very large or cause trouble. Mediastinal emphysema may follow injury to the lung or bronchi. By pressure on the structures in the mediastinum this may cause dangerous symptoms. While this condition is difficult to control, it can sometimes be retarded by making an incision in the suprasternal notch to allow for the escape of air.

CONCLUSIONS

The extensive use of aerial warfare is making every doctor a potential military surgeon. Correctly to handle war wounds of the chest, the physiology of the chest and the forces that cause these wounds must be understood.

Shock, open pneumothorax, tension pneumothorax and hemothorax, and cardiac tamponade must be handled promptly if the lives are to be saved.

Some of the newer ideas in the treatment of these wounds are discussed.

BIBLIOGRAPHY

Bailey, Hamilton: Surgery of Modern Warfare, Baltimore, Williams & Wilkins, 1941.

Berry, F. B.: Wounds of thoracic viscera, Am. J. Surg., 39:12, 1938.

Bigger, I. A.: Heart wounds; report of 17 patients operated upon in Medical College of Virginia hospitals and discussion of treatment and prognosis, J. Thoracic Surg., 8:239, 1939.

Blades, B.: Emergency treatment of traumatic chest injuries, S. Clin. North America, 20:1473, 1940.

Boland, F. K.: Chest wounds in military and civil practice, Mil. Surgeon, 81:175, 1937.

Coller, F. A., and Farris, J. M.: Surgery of modern warfare, Surg. Gynec. & Obstet., 72:15, 1941.

Conklin, F. L.: Symposium on military surgery; surgery of chest, Surg. Clin. North America, 21:1593, 1941.

Crafoord, C.: Gunshot wounds of lungs, Nord. Med. (Hygiea), 3:2727, 1939.

Dean, D. M., Thomas, A. R., and Allison, R. S.: Effects of high explosive blast on lungs, Lancet, 2:224, 1940.

DeBakey, M.: Management of chest wounds; collective review, Internat. Abstr. Surg., 74:203, 1942.

Dickson, D. E.: Surgical problems of war; more notes on penetrating chest wounds, Brit. M. J., 1:403, 1940.

Edwards, F. R., and Davis, H. M.: Traumatic haemothorax, Lancet, 2:673, 1940.

Frey, E. K.: Ueber Lungenschusse, Munchen. med. Wchnschr., 87:1197, 1940.

Fulton, J. F.: Blast and concussion in present war, New England J. M., 226:1, 1942.

Gordon-Taylor, G.: Problems of surgery in total war, with special reference to abdominal injuries, Surg. Gynec. & Obstet., 74:375, 1942.

Hadfield, G., and Christie, R. V.: Case of pulmonary concussion ("blast") due to high explosive, Brit. M. J., 1:77, 1941.

Hadfield, G., et al.: Blast from high explosive: preliminary report on 10 fatal cases, with note on identification and estimation of carboxyhaemoglobin in formol-fixed material, Lancet, 2:478, 1940.

Harmon, P. H., Baker, D. R., and Kornegay, R. D.: Uncomplicated fractures of ribs and major injuries of chest wall; treatment by infiltration with local anesthetic, J. A. M. A., 118:30, 1942 .

Hegner, C. F.: Surgical treatment of injuries of chest, Am. J. Surg., 47:394, 1940.

King, J. D., and Curtis, G. M.: Lung injury due to detonation of high explosive, Am. J. Surg., 74:53, 1942.

Mitchiner, P. H., and Cowell, E. M.: Air-raid; chest wounds, Lancet, 1:534, 1939.

Monod, O. M.: Lobectomy in case of injury of hilum of lung; report of two cases, Thoracic Surg., 10:475, 1941.

Monod, R.: Quelques retouches aux directives admises du traitment des plaies penetrantes de poitrine, Press med., 48:737, 1940.

Moorhead, J. J.: Surgical experience at Pearl Harbor, J. A. M. A., 118:712, 1942.

Moorhead, J. J.: Personal communication.

Ranson, F. T.: Notes on gunshot wounds of chest, J. Thoracic Surg., 9:79, 1940.

Roberts, J. E. H., and Tubbs, O. S.: Recent experience with war wounds of chest, Am. J. Surg., 54:289, 1941.

Steinke, C. R.: Penetrating gunshot and stab wounds of thorax; report of 87 cases, J. Thoracic Surg., 8:658, 1939.

Thomas, C. P., et al.: Discussion of chest injuries, Proc. Roy. Soc. Med., 34:83, 1940.

Trueta, J.: Organization of hospital services for casualties due to bombing of cities, based on experience gained in Barcelona, with special reference to classification of casualties, Proc. Roy. Soc. Med., 33:13, 1939.

Zuckerman, S.: Experimental study of blast injuries to lungs, Lancet, 2:219, 1940.

Zuckerman, S., et al.: Discussion on problem of blast injuries, Proc. Roy. Soc. Med., 34:171, 1941.

DISCUSSION

Dr. Frederick F. Boyce (New Orleans): The statistics which Dr. Harrison has presented concerning the frequency and the serious consequences of war wounds of the chest are evidence of the importance of their consideration at this special time. I am sorry that the limited time at his disposal did not permit him to discuss in detail all the complications which follow such wounds. The time at my own disposal is even more limited, and I shall therefore merely comment on one or two special types of wounds.

Colonel Yates, who was in charge of the American unit detailed in the last war to study the treatment of thoracic injuries, was an advocate of immediate open operation in the management of all but the most limited degree of hemothorax. His

views were in direct opposition to those of Morelli, who advocated aspiration and air replacement, on the ground that air is an irritant to serous membranes and that the first principle in the prevention as well as the treatment of pleuritis is the elimination of irritation. It is interesting to observe, in special articles and surveys in the recent British literature, that Morelli's method now seems to be the treatment of choice in severe injuries of this kind. In lesser degrees of tension pneumothorax the use of valvular trocars, which permit the egress of air but prevent its ingress, is reported to be as satisfactory as repeated aspiration.

Several rather ingenious methods have been developed for the treatment of sucking wounds. In the last war the Italians used a dumbbell shaped rubber bag which was inflated after its insertion into the wound and by which closure was thus effected. Tiegel devised a valve drainage plate which, when applied to the wound, served for drainage as well as effected closure and thus guarded against the development of hemothorax and tension pneumothorax.

When mediastinal emphysema develops, it would seem more logical, instead of making an incision in the suprasternal notch, which was formerly the custom, to attack the problem at its source. Aspiration of the pleural cavity or water-sealed drainage is the best plan when there is a tear or laceration of the pleura. Operative intervention, with closure of the defect, should be considered when injury to the bronchus is suspected.

The best treatment of chylothorax due to injury of the thoracic duct is the use of supportive measures, with aspiration to relieve cardiorespiratory embarrassment, since surgical repair is impractical. If the aspirated chyle is sterile, it could be reintroduced into the vein.

Finally, sulfonamide therapy should supplement whatever other methods of treatment are employed. In a recent study of war wounds of the chest, Roberts and Tubbs showed that following the prophylactic use of sulfanilamide and its derivatives there was a decrease in the incidence of infection, and, when infection did occur, a decrease in the severity of the toxemia which is always a grave phase of wounds of the chest.

Dr. Frank L. Loria (New Orleans): I think we should be grateful to Dr. Harrison for bringing this subject to our attention, since it is so timely. The time is at hand when every physician, irrespective of his specialty, should become more or less surgically minded. In our present national crisis one can never know when a tragic situation will occur needing his services, be he surgeon or internist.

My interest in chest injuries has come about because of the frequent association of injuries to this cavity with abdominal injuries. Two days ago I presented a paper, before this section, on abdominothoracic gunshot injuries, reviewing statistically the cases admitted to the New Orleans Charity Hospital for 15 years prior to 1942.

Chest injuries require a consideration of four important factors: (1) hemothorax; (2) hemopneumothorax; (3) open or suction pneumothorax; (4) valvular or tension pneumothorax. Any of these factors may be sufficiently severe to cause death; and most deaths from chest injuries, aside from those caused by injury to the heart or other important mediastinal structures, usually are the result of one or more of the above mentioned factors.

Hemothorax rarely results from injury to the lung alone. The systolic blood pressure in the pulmonary circulation is only about 20 mm. of mercury, just about one-sixth of what it is in the general circulation. In addition the lungs are credited with certain hemostatic properties. Thus any pulmonary hemorrhage is usually not of long duration nor very great. Hemothorax usually results from injury to the intercostal or the internal mammary vessels. The presence of a large hemothorax should therefore direct the surgeon's attention to these structures.

As the time limit for this discussion is rather brief I shall have to pass over the three other important complications of these injuries, particularly since I wish to present certain figures in connection with a study of chest injuries I am now making on those cases admitted to the New Orleans Charity Hospital over a period of 15 years prior to 1942. During this period 304 cases of penetrating gunshot wounds of the chest were admitted to this institution with 106 deaths, giving a mortality rate of 34.83 per cent. In this same period of time among 628 cases of penetrating stab wounds of the chest there were 73 deaths, giving a mortality rate of 11.6 per cent. The great difference in the mortality among these cases, caused by different weapons, is no doubt the result of the more frequent occurrences of the above named important complications in gunshot injuries over stab wounds of the chest.

Dr. Isidore Cohn (New Orleans): We have with us five representatives of the government hospital here in New Orleans. I am wondering if Colonel Woldenberg of LaGarde Hospital would have a few words to say.

Lt. Col. S. C. Woldenberg (New Orleans): I am rather astonished to be asked to say something. First of all I came in to the meeting late and I am sorry I missed some of Dr. Harrison's paper.

In the closing part of the paper Dr. Harrison indicated that he still reduces fractures of the rib. At LaGarde Hospital we have not received any actual war casualties yet. We have a lot of other problems of chest cases confronting us in the surgical department. Empyemas, for instance, following pneumonias, encapsulated abscesses and cysts, which are similar to the cases seen in civilian practice. When receiving a chest injury case, we are not immediately confronted with the necessity of reducing the fractures of the rib. The primary importance, if the patient is in shock, is to stop

the hemorrhage and give him supportive measures, transfusions, infusions, stimulation, or whatever may be needed. In general, we are primarily confronted with whatever emergency measures are necessray to save the patient's life. Afterwards, when the patient has sufficiently recovered, we then start on a definite course of treatment for the specific injury.

Dr. Harlan W. Harrison (in closing): In regard to Colonel Woldenberg's statement about fractures, I am afraid I did not make this point clear. I meant reduction of the fractured rib should only be carried out when there is that fracture of the rib which is penetrating into the lung or causing severe intracostal bleeding.

In regard to the hemothorax, that is probably one of the most common conditions in treatment of chest injuries. I think the work of Weeks of the New York police force is interesting in which he reported 76 cases of gunshot wounds of the chest were treated with late or no aspiration, with a mortality of 26 per cent; subsequently he started aspiration on all hemothorax cases early, within 24 or 48 hours and in a series of 17 cases there was no mortality.

I am sorry I could not completely cover the subject as I had planned. In closing I shall emphasize control of hemorrhage, control of shock and in an emergency close up any sucking wounds which cause definite disruption of the normal physiology.

—o—

SURGICAL TREATMENT OF PELVIC THROMBOPHLEBITIS*

LIGATION OF INFERIOR VENA CAVA AND OVARIAN VEINS

A PRELIMINARY REPORT

CONRAD G. COLLINS, M. D.†
JACK R. JONES, M. D.†
AND
EDWARD W. NELSON, M. D.†
NEW ORLEANS

The high mortality associated with pelvic thrombophlebitis, the relative inefficiency of chemotherapy and other conservative methods in this condition and the poor results obtained by ligation of the ovarian veins alone prompted the authors to adopt a more radical type of operative procedure, in an attempt to better previous results.

*Read before the Orleans Parish Medical Society, May 11, 1942.

†From the Department of Obstetrics and Gynecology, Tulane University and the Tulane Unit, Charity Hospital, New Orleans, Louisiana.

A study of a series of cases of pelvic thrombophlebitis in the female[1] found at autopsy or operation show that this condition is not infrequently found in postpartal and postabortal sepsis. Occasionally it follows pelvic surgery, cervical stricture, or the application of radium. Metastatic emboli are common and patients who do survive under a conservative regimen survive only after a prolonged convalescence.

In this communication, we wish to present three cases of pelvic thrombophlebitis that have recently come under our observation. All three were treated by ligation of the ovarian veins and the inferior vena cava.

CASE No. 1

M. N., a 22 year old colored primipara, delivered by low forceps and episiotomy a 7 pound, 6½ ounce viable male baby from L. O. T. position after a 17 hour, 40 minute, fairly difficult labor, on February 5, 1942. She had a non-morbid course until seventh postpartal day when the temperature then rose to 101°. The next day, February 13, there was a chill with increased temperature to 104.4°.

Vaginal examination on February 14: Lochia, not foul; episiotomy, well healed; cervix closed with a healing laceration which extended to right fornix; slight parametrial tenderness. The uterus was not tender, and well involuted; adnexae were negative. Chest, essentially negative, clinically. Urinalysis, 10 pus cells per high powered field; residual urine 10 c. c. Blood transfusion 200 c. c.

On February 15 she had a chill and temperature was 104°. Blood: hemoglobin, 60 per cent, white blood cells 19,600, polys 86 per cent. Urinalysis, negative. Blood culture taken (reported negative on February 20). Sedimentation rate, 20 minutes.

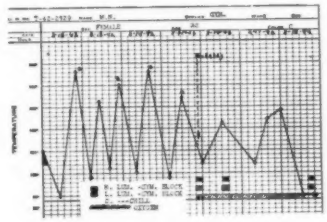
Chest clinically negative. X-ray: "Bronchopneumonic infiltration in lower two-thirds of the right lung with multiple small cavities present; the process could be due to pneumonia, with abscesses, local tuberculosis or multiple septic infarcts, which are microscopic in the left lung."

Recto-vaginal examination: Definite tender thrombi in both broad ligaments about level of uterine plexus. Diagnosis: Pelvic thrombophlebitis.

Operation: Under 20 mgm. procaine spinal anesthesia, ligation of right ovarian vessels one to two inches from their junction with the vena cava and resection of vessels with right tube and ovary. Ligation of inferior vena cava one inch proximal to junction of common iliac veins and ligation of left ovarian vessels above pelvic brim.

Pathology found at operation: (1) Thrombi at hilum of right ovary; (2) thickening of uterine veins; (3) thrombosis in right iliac vein.

The patient's postoperative course and the therapy administered postoperatively are indicated in Chart 1.



X-ray examination of chest postoperatively showed the following: February 18: "Infiltration of both lungs more marked on right, with what appears to be multiple small cavities on the right side. There is elevation of the left leaf of the diaphragm and disc-like atelectasis." February 24: "Slight clearing of the infiltration in the right hemothorax." March 1: "Clearing of right lung field since last examination. The left leaf of the diaphragm is still elevated. There is also clearing of the left lung field." March 19: "Complete clearing of the infiltration in both lungs, there is still elevation of the left leaf of the diaphragm."

This patient has recovered completely and remained well to date.

CASE No. 2

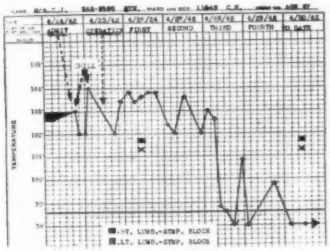
Mrs. C. J., aged 27, had chills, fever and vaginal bleeding. L. M. P. February 25, 1942, for 5 days. P. M. P. January 24, 1942, for 5 days. In March she took large doses of quinine, and again on April 2, 1942, but did not bleed. On the afternoon of April 17, 1942, she fell and injured herself, and at 2:30 p. m. April 18, 1942, she had a light chill and fever. Since that time, she has had many chills during which time her teeth would chatter and she would shake the entire bed. Following these chills, she would have an elevation of temperature to 105° by thermometer. Chills have continued to become increasingly worse until the day before admission, when she began to pass blood. She has suffered with nausea but has not vomited. She denies an attempted criminal abortion.

She had rheumatic fever as a child; no edema of ankles; dyspneic at times; no precordial pain; cough for eight months; no hemopytsis. Gastrointestinal history negative. She had slight dysuria, but no hematuria.

Physical examination: B. P. 108/72. Head: Negative. Mouth dry, membranes pale. Neck: Negative. Chest: Heart not enlarged; no thrills; systolic murmur at pulmonic area and aortic area; diastolic murmur at mitral area. Abdomen: Negative. Genitalia: Multiparous, relaxed perineum, cervix blue, soft, lacerated, necrotic, and dilated 1 cm. There is a purulent material coming from the cervix. Bimanual examination showed the presence of thrombotic veins in the right parametrium.

Diagnosis: Septic abortion, rheumatic heart disease, pelvic thrombophlebitis. X-ray April 24: "No evidence of pneumonia."

Operation on April 25: Novocain spinal 200 mg. Procedure: Ligation of right and left ovarian arteries and vein. Ligation of inferior vena cava at lower level of fourth lumbar vertebra. Operative finding: Thrombophlebitis of both uterine veins.



This patient's postoperative course and therapy administered are indicated in Chart 2. She has remained well to date and at present the pregnancy is following a normal course.

CASE No. 3

Mrs. C. J., aged 67, had a vaginal hysterectomy, anterior and posterior colporrhaphy three weeks ago, followed by an uneventful recovery. She was ambulatory on third day following operation. Discharged symptomless, but running a low grade fever.

Readmitted March 24, 1942, 11 days later, with history of having fever and having contracted a "cold." Pain in left flank for several days, but not present on admittance. Patient states she had chills and fever to 103° while at home, and dysuria several days. All these symptoms subsided and temperature was normal on admission.

For three days after admission this patient was perfectly well and afebrile. Urinalysis 1-2 pus cells per high powered field; residual urine 20 c. c. on March 25. Residual urine March 26, 6 c. c. At this period we began to doubt her history; however, on the night of the third day in the hospital she had a chill and elevation of temperature to 104°.

On March 26, p. m., urinalysis was negative. On March 27, urinalysis was negative; blood culture was negative; x-ray of chest was negative; sputum was negative; blood count: red blood cells 3,940,000, white blood cells 7,850, polys 75 per cent. At this time a diagnosis of pyelonephritis, bronchopneumonia, and possible thrombophlebitis was entertained. X-ray and urinalysis and clinical aspects ruled out. Later in day there was noticed some tenderness of thighs. A malaria smear was negative. Sulfathiazole was increased to full therapeutic doses.

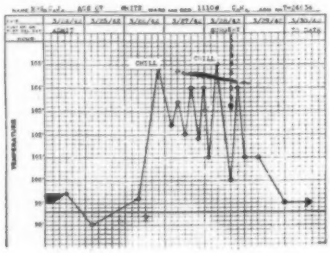
On March 28 she had another chill and increased temperature to 105°. A diagnosis of pelvic throm-

bophlebitis was more apparent and venous ligation was decided upon. G. U. consultation and cystoscopic done. Smear and culture from bladder, left and right kidney was negative.

Portable x-ray: "Infiltration in both lungs, more marked on the right; this could be due to pneumonia or to small infarcts." White blood cells 7,800.

Operation on March 28: Local infiltration 1 per cent procaine. Procedure: Ligation of vena cava at level of fourth lumbar vertebra.

The patient has recovered completely. Her postoperative course is shown on Chart 3.



DISCUSSION

Three cases of thrombophlebitis of the pelvic veins treated by ligation of the ovarian veins and the inferior vena cava are presented: One case was postpartal, one followed an attempted criminal abortion, and another followed a vaginal hysterectomy. All cases have fully recovered and at present do not show any ill effects from vena cava ligation. True, the venous pressure in the lower extremities of all three is elevated at present but there is no edema. The venous pressure has shown a decrease in all cases as the time interval from date of operation increases. This leads us to believe that in the near future the venous pressure will return to, or vary little from the normal. Dr. George Burch, of the Department of Medicine, Tulane University, is studying the circulatory system in these cases at regular intervals and will publish a separate report on his findings at a later date. It is exceedingly interesting that one patient (case 3) is 67 years of age and another (case 2) had rheumatic heart disease, and no ill effects have been observed following ligation of the inferior vena cava.

All cases of vena cava ligation should have bilateral lumbar sympathetic blocks following the operation. This enhances the development of collaterals and relieves any edema that might be found postoperatively. Occasionally a patient will feel faint and anxious while the block is being administered. Should this be the case, the procedure should be discontinued immediately and attempted again in 48 hours. In the cases presented there is no visible edema at present, no matter how long the patient remains ambulatory. In all cases the pulmonary complications have cleared completely and no one has shown any recurrence of fever.

In none of these cases was chemotherapy administered postoperatively and therefore any improvement in the patient's condition cannot be attributed to this mode of therapy.

The preoperative diagnosis is occasionally extremely difficult to determine with certainty. In these three cases the preoperative diagnosis was made by the senior residents, corroborated by the senior author. Operation was performed by Drs. Jones and Collins in case 1, Drs. Warren and Collins in case 2, and Drs. Thomas and Holmes in case 3.

CONCLUSIONS

1. The possibility of thrombophlebitis of the pelvic veins must be considered in postabortal or postoperative cases showing septic temperature associated with chills. X-ray examination of the chest in all cases is imperative as emboli and infarction may be present without clinical symptoms.

2. Careful examination of the urinary tract, including cystoscopy, should be performed in order to eliminate this system as the causal agent in the production of the septic curve and chills. This procedure was followed in all of our cases.

3. Should patients with septic temperature and chills fail to respond to chemotherapy, then surgical therapy should be instituted.

4. Thrombophlebitis and phlebothrombosis of the veins of the lower extremities should be searched for. Phlebograms are of definite value in proving or disproving the presence of these conditions.

5. The surgical procedure should consist of:

a. Ligation of ovarian veins and hypogastric veins if thrombi are found in the ovarian veins and not the uterine, hypogastric or iliac veins.

b. Ligation of ovarian veins and inferior vena cava if thrombi are found in the ovarian veins and in the uterine, hypogastric or iliac veins.

c. Ligation of the ovarian veins and inferior vena cava if no thrombi are found in the ovarian veins but are found in the uterine, hypogastric or iliac veins.

In this manner the normal venous channels from the uterus are ligated.

6. The finding of thrombosed pelvic veins on vaginal examination is a distinct aid in diagnosis. However, failure to find them by palpation does not mean that thrombophlebitis of the pelvic veins does not exist.

REFERENCE

1. Nelson, E. W., Jones, J. R., and Collins, C. G.: Pelvic thrombophlebitis: A study of the etiological factors from a statistical standpoint, New Orleans M. & S. J. (in press).

DISCUSSION

Dr. G. E. Burch (New Orleans): We have been conducting studies of circulation in these patients. It is not surprising that these patients do not develop edema. It is practically impossible to produce edema of the legs of dogs by ligation of the vena cava. Three years ago Dr. DeBakey and I studied the influence of sympathetics in phlebitis. All animals were found to develop a marked increase in venous pressure distal to a ligated femoral vein. There was a simultaneous extensive drop in the volume of pulsation of the small peripheral vessels. The non-involved vessels pulsated normally. This is very much like observations found in the finger tip when the upper extremity is elevated or lowered; in the former position there is great increase of volume pulsation of the peripheral blood vessels and in the latter position there is a decrease in pulsations. In the patients in whom Dr. Collins ligated the inferior vena cava, it was found that the venous pressure increased quite markedly in the lower extremities. In some of these patients, followed over a period of three weeks, it was found that there was gradual reduction in this venous pressure. In no instances did pressure return to normal. As Dr. Collins said, it will take many weeks probably for that to happen. As found in dogs, there was quite a marked decrease in pulsations of the peripheral blood vessels in the toes while those in the fingers were normal.

One wonders why ligation of the inferior vena cava is so beneficial. One might conjecture an explanation. When infection begins in tissue, there is first an accumulation of antibodies at the site of infection, so as to destroy the offending organism. A limiting wall is also built to prevent spreading. If infection occurs in a vein there is continual washing away of cellular and humoral immune bodies, so that a sufficient concentration cannot accumulate to overcome this infection and to isolate it locally. With ligation of the veins the circulation is slowed or stopped, so that it is possible for these antibodies to accumulate in sufficient quantities to isolate and overcome the infection.

Dr. Curtis Tyrone (New Orleans): This presentation represents an epoch in the treatment of this type of pevlic infection. Dr. Collins and his associates are to be commended for their study and ingenuity in carrying out this operation and should be congratulated on the results obtained. I can remember the time when any form of venous ligation for this type of pelvic infection was considered radical and we approached with a great deal of fear and in many patients in whom it was tried it was a failure. In the light of Dr. Collins' work here, probably the reason for failure was that we did not go far enough and ligate the veins more extensively as he has done.

Dr. Nelson's presentation certainly presents an interesting statistical study. I think he has gone into it very thoroughly and one thing pointed out here is that when there is thrombophlebitis, which develops postoperatively, and pelvic infection, it usually is in some case where operation has been done on the cervix or an upper vaginal hysterectomy has been done.

Another interesting conclusion these statistics brought out is that the mortality, which we know is 100 per cent in pelvic thrombophlebitis without operation, has been reduced to 12 per cent.

Dr. E. L. King: This carries us back to the work of Dr. Jeff Miller. In 1912 at the A. M. A. Dr. Miller reported all he could find in the literature to that date regarding this operation for pelvic thrombophlebitis. At times he waxed enthusiastic and at times he did not. In those days we waited too long in many cases to establish diagnosis and waited still longer, hoping that whatever conservative method of treatment was employed would accomplish results. Finally in desperation the operation was performed and it would be unsuccessful. However, I did see him accomplish very wonderful results in private cases mostly referred, ligating not the vena cava but one or the other of the iliacs, depending on the involvement. On one occasion the ureter happened to be wounded and the kidney had to come out. The patient was saved by the operation.

One thing which should be brought out is that blood cultures in these cases are not satisfactory.

I believe that is due to several factors. One is time that the culture is taken. We learned years ago that the best time to take culture is at or after chill. It is not always easy to get the technician to come immediately at the time of chill. Another thing is that not enough cultures were made anaerobically; a great many infections are due to anaerobic organisms.

Dr. Collins mentioned that we can not often feel these veins. This is put down as a classical method of diagnosis. He mentioned only one case in which they were felt; only once in a great while can they be found. Diagnosis has to be based on the clinical picture—chills, up and down temperature and history of gynecologic or obstetrical operation or manipulation. Early diagnosis is important, based on the clinical picture. We must not get too enthusiastic and should not ligate every vena cava in everyone with fever after pelvic operations or delivery. At the same time it has been shown conclusively that it can be done without killing the patient; collateral circulation is established and the patient will usually get well.

Dr. Miller used to feel that by tying off the vein you jugulated the infection; that the bacteria did not go through the vein wall but there was steady crumbling of the thrombi. The thrombus in the vein would break down and produce a showering of emboli throughout the body. By ligating the vein above the thrombus it was jugulated and tied up and there was no more shower and then the antibodies would work and kill off the bacteria.

It is certainly of great interest that so many of the patients have been cured. So far this treatment is the only thing we have found of real value for these cases of thrombophlebitis.

Dr. B. B. Weinstein (New Orleans): I think Drs. Collins, Nelson and their associates are to be commended on the excellent work they have done, and on the fine presentations they have given us. One more greatly appreciates the value of their report when we realize that this procedure, looked upon by many as radical, proves to be not only life saving, but conservative with a 12 per cent gross mortality and a corrected mortality of zero in Collins' series. I am interested in the fact that the ligation of the vena cava is relatively so safe, and so little peripheral edema is evident postoperatively. I think one of the explanations of this finding is the very rich venous collaterals set up after ligation of the inferior vena cava. These collaterals can be divided largely into three groups: (1) superficial, composed of collaterals set up via the superficial epigastric, and superficial circumflex iliac veins, which thus connect the saphenous flow from the lower extremities with the superficial thoracic and superficial upper abdominal wall venous drainage; (2) deep, composed of collaterals set up via the anastomoses of the deep (inferior) epigastric and deep circumflex iliac veins with the superior epigastric and lumbar veins, thus connecting the external iliac veins with the internal mammary veins and the ascending lumbar trunk and finally (3) the ascending lumbar trunks which form a most important part of the venous collaterals after ligation of the vena cava. These trunks begin on either side of the sacral-promotory where they communicate with the middle and lateral sacral veins, common iliac hypogastric and ilio lumbar veins via the anterior sacral plexus. They ascend in front of the transverse processes of the lumbar vertebrae and communicate with the lumbar veins, the vena cava inferior and the right renal vein. The right trunk continues as the azygosi vein to enter into the superior vena, the left continues as the hemazygos which ultimately drains into the azygos vein.

Dr. M. E. DeBakey (New Orleans): Dr. Collins and his associates deserve special commendation for their zeal and determination in attacking such a serious problem and their courage in directing attention again to the value of a procedure which on first thought may seem radical but upon more sober consideration has a definitive rationale. Before performing his first case Dr. Collins discussed with me the possibility of vena caval ligation and at that time it was recalled that the procedure had been successfully performed a number of years ago and was apparently indicated in the particular case under discussion.

Proximal ligation in venous thrombosis has an interesting historical development. Perhaps the earliest realization of the rationale of this procedure was by Hunter, who, in 1784, successfully applied it clinically. However, Hunter apparently did not actually perform venous ligation but attempted to accomplish the same purpose by placing compression "upon that part of the vein just above the suppuration." Possibly Lee, in 1865, was one of the earliest actually to ligate a vein above the thrombophlebitic process. About 25 years later Kraussold recorded the successful ligation of the femoral vein in a patient with suppurative thrombophlebitis following thigh amputation and Zaufal reported the successful ligation of the internal jugular vein in the treatment of pyemia originating in the internal ear. It would seem that the earliest surgeon to apply the method in gynecology was Freund, who, in 1898, performed ligation and excision of the thrombosed ovarian vein and broad ligament in two cases but the procedures were unsuccessful. Perhaps Trendelenburg, in 1902, was the first to perform the procedure successfully in a case of puerperal infection. At the first operation he ligated the hypogastric vein but because the patient continued to have chills he performed a second operation and ligated the ovarians. It is interesting to observe that at that time Trendelenburg expressed the opinion that vena caval ligation should not be considered because if thrombosis was that extensive the procedure would not be successful. Nine years later, Trendelenberg had

apparently changed his mind as he reported probably the first successful case of vena caval ligation. Since then the procedure has been performed by a number of surgeons. In 1937, Krotoski collected 48 cases and stated that there was no evidence of subsequent circulatory disturbances in the lower extremities. Krotoski also directed attention to the suggestion of Schroeder and Klauberg that because of the frequent difficulty of finding a thrombus, the vena cava and the ovarian veins should be ligated in all cases.

Apparently proximal venous ligation in gynecologic cases received greatest enthusiasm about 20 to 25 years ago. The procedure was very popular in Germany and there was heated discussion and much controversy concerning the respective values of the transperitoneal and extraperitoneal approaches and whether the operation should be done after the first or the third chill. In this country Dr. C. Jeff Miller's exhaustive consideration of the subject will be recalled by many in the audience. Gradually the pendulum swung toward conservatism and the operation began to be considered dangerous and of little or no value. This was due to the appallingly high mortality and the poor results observed by numerous surgeons. In his critical analysis of the subject Krotoski justifiably directs attention to a number of factors which may have accounted for these results, the most important being that the operation was frequently performed too late, by inexperienced surgeons, and not sufficiently extensive. It is gratifying to see the recent revival of interest in this procedure which undoubtedly has a definitive place in the management of these cases.

The only other thing on which I wish to comment is the value of phlebography. While it is undoubtedly significant in the type of cases described by Dr. Collins, it is of even greater importance in certain forms of phlebothrombosis in which the condition is suspected but not clinically demonstrable. Such a case was recently observed. The patient was first treated for pneumonia and suspected of having heart disease. Subsequently the latter was excluded and it was realized that the patient was suffering with pulmonary infarcts and phlebothrombosis was suspected although there was no other clinical evidence of the condition. Dr. Ochsner, who was called in consultation, performed phlebograms in both lower extremities and these demonstrated clearly the presence of normal venous channels in one extremity and venous thrombosis in the other. Following proximal venous ligation in the latter the patient's temperature dropped to normal and a rapid recovery ensued.

Dr. Edward Nelson (In closing): An important fact was brought out by Dr. DeBakey, namely, that in many of our cases it would have been possible to make a different diagnosis, preoperatively. This is easily explained. The postpartal cases frequently have complicated deliveries, often followed by urinary retention. This predisposes to pyuria, and often the fever is carelessly blamed on urinary tract infection. In our cases, we found it necessary to subject the patients to cystoscopic examination, in order to rule out the urinary tract as the source of fever.

Pulmonary infarction, on the other hand, is too often confused with pneumonia. In reviewing the records, it was found that in all but a few instances, a clinical diagnosis of pneumonia was made, and the patients did not have the benefit of an x-ray examination. Since we started this work, the Department of Radiology has concentrated more on the diagnosis of pulmonary infarction. Since the lesions are so often atypical, studies are now being conducted to compare them, and determine the frequency of the different types.

The remark made, that it takes courage to operate on these patients, is certainly true. The diagnosis is often not definite, but the patients must be given the benefit of the doubt, especially when they are not responding to a conservative regimen, and an exploration of the pelvic veins conducted.

Dr. Conrad Collins (in closing): Ligation of the vena cava was not attempted until I talked over the possibilities and possible complications with Drs. Gage, DeBakey, Ochsner and Rives.

I wish to give credit to the resident staff on the Tulane Unit, Charity Hospital division of gynecology and obstetrics for their early diagnosis in these cases.

In regard to presence or absence of thrombi, in one patient on whom Dr. Rives and I operated, at the first operation we found thrombi in the ovarian veins and they were ligated and segments were removed. The patient was well for a week and then had more chills and fever. At the second operation, performed three weeks later, we were unable to demonstrate any other thrombi in the ovarians above the ligature or in the iliacs, hypogastrics or uterine veins, but the vena was ligated anyhow. The patient recovered completely.

—o—

THE USE OF THE DARKFIELD MICROSCOPE FOR DIAGNOSIS OF GENERALIZED SECONDARY LESIONS OF SYPHILIS

OWEN F. AGEE, M. D.
NEW ORLEANS

Of course, the use of the darkfield apparatus for identification of the *Treponema pallidum* in primary lesions of syphilis, genital or otherwise, and in moist papules, particularly about the genitalia, is generally recognized as good diagnostic procedure. The presence of syphilis organisms in other types of skin lesions might be generally ad-

mitted by many individuals who would not stop to consider the advisability of searching for the organisms.

Vryonis and Morgan[1], in 1939, made a study of the numerical relationship of *Spirochaeta pallida* in the various types of early syphilitic lesions. In this article they placed the lesions of syphilis in the number of organisms in the following order:

	Average No. Spirochetes per cu. mm.
1. Condyloma	37,620
2. Mucous patch	24,700
3. Chancre	14,700
4. Dry secondary lesions	1,800

The dry secondary lesions ranged from 370 to 3,200 in the number of spirochetes per cubic millimeter.

Naturally the use of the darkfield microscope in secondary syphilis is not absolutely necessary in order to diagnose the disease as syphilis, for in this stage of the ailment the serologic tests are almost universally positive. But if one wishes to say with absolute certainty that he is dealing with secondary syphilis, it would seem that he should attempt to use the darkfield for proving the lesions. The presence of positive serologic tests and the presence of skin lesions suspected of being syphilis do not necessarily prove the case for secondaries. Furthermore, the beginning of treatment can be expedited by obtaining a positive darkfield report instead of waiting for a positive serologic report.

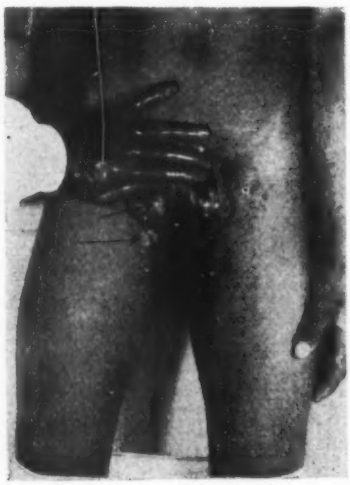


Fig. 1 (case 1). Shows condylomata of thigh and scrotum, both of which were positive for *Treponema pallidum*.

The following cases, eight of which were seen in the New Orleans Health Department Clinic and two in the Tulane Clinic at Charity Hospital, illustrate the practical use of darkfield apparatus in either dry or closed secondary lesions:

CASE REPORTS

No. 1. B. K., a colored male, aged 23, was an itinerant stable boy. This patient was seen with condylomata of the anus, scrotum and intertriginous regions of the thighs. He had annular lesions also, of the face and neck. Darkfield examinations of material were done as follows:

Condyloma of thigh.....positive
Condyloma of scrotum.....positive
Annular lesion of neck.....positive

The patient began treatment on the day of examination. The serology proved to be positive.

No. 2. A. Mc., a white female, aged 31, was named by the military as a contact and probable source of a case of primary syphilis. On the day that she was examined it was noted that she had a generalized maculo-papular rash, allegedly of one weeks' duration. There was some desquamation. A questionable mucous patch of the mouth was noted. One of the largest scaling lesions of the neck was selected for darkfield examination. The top was scraped away and from the serum beneath a positive darkfield was obtained. The blood proved to be positive. The patient began treatment on the day of the examination. Incidentally the darkfield examination of the mouth lesion was negative.

No. 3. B. M. F., a white female, aged 21, had been named by the military as a contact of a case of gonorrhea. When she appeared for examination she had a mucoid vaginal discharge and a maculopapular rash of the abdomen, arms, legs and chest. A fairly large closed papule of the abdomen was chosen for a darkfield examination. The top was scraped away and from the serum beneath a positive darkfield was obtained. The blood proved to be positive. The patient began treatment on the day of examination. Her common-law husband came in for examination three days later and at the various other times as shown below with negative results as follows:

February 23, 1942—Negative examination, negative blood.

March 3, 1942—Negative examination, negative blood.

March 18, 1942—Negative examination, negative blood.

On April 11 he was again examined. At this time he was found to have a penile lesion of three days' duration. Darkfield examination of the lesion was positive; the serologic test was negative. He began treatment that day.

Fig. 2 (case 1). Shows annular lesions of the neck, one of which was positive for *Treponema pallidum.*

No. 4. O. O., a colored male, aged 30, came to us with a history of having had a rash for the past month. He had an old granulating mass of the pubic region which allegedly had been present for two months. There were about twenty papules of the chin, varying in size from two to four mm. in diameter, some of which were umbilicated. The top was removed from the largest lesion of the chin and the serum was examined by darkfield. It was positive, (loaded). The serologic test was positive. Treatment was begun on the day of the darkfield examination.

No. 5. A. R., a white male, aged 57, was referred to us on June 20, 1942 because he had a penile lesion, a rash, and a positive serologic test. We saw him on June 24, 1942, at which time he had a large dirty crusted lesion of the penis, involving much of the glans and coronal sulcus. An attempt at darkfield examination was not made on the penile lesion. It had been present two months, according to the history. There were scattered papules of the face, abdomen, arms and legs. A fairly tense shiny papule of the arm was chosen for darkfield examination. The surface was abraded and the exuding serum examined. It was positive for *Treponema pallidum.* In this case we might have been hesitant about making a diagnosis of secondary syphilis without benefit of a positive darkfield. We would have considered that at the age of 57 the man must have had many opportunities to have positive serology from previous escapades.

No. 6. M. C., a colored female, aged 20, had been hospitalized on May 23, 1942, for a rash of six days' duration, with coryza, and other symptoms of measles. She was suspected of having bronchopneumonia, and a drug rash as well, since she had had sulfathiazole. Her serology was positive, and a diagnosis of secondary syphilis was made. Allegedly she had had a sore of the genitalia three weeks before going to the hospital. The patient was referred to us June 6, 1942, at which time she had a generalized scaling papular rash. The darkfield examination of a large papule of the dorsum of the foot was positive, proving that she actually did have secondary syphilis. Her serology was again positive.

No. 7. Mrs. O. E., a white female, aged 25, had been seen in the dermatology clinic with a diagnosis of pityriasis rosea. Serology was positive and she was referred for treatment. At the time of referral she had an insignificant rash of the chest, consisting of a few scattered papules. The patient insisted that it was a different eruption from the pityriasis she had had at first. A closed papule over the sternum was chosen for darkfield examination. The top was abraded and the pulp from beneath, mixed with a tiny drop of normal saline, was examined by darkfield. It was positive for *Treponema pallidum.*

No. 8. Mrs. E. A., a white female, aged 55, was found to have a positive serologic test in the Dermatology Clinic. When she was referred for treatment she was found to have a thick papular erup-

Fig. 3 (case 2). Shows maculo-papular lesions of the neck, one of which was positive for *Treponema pallidum.* (Lesion used for darkfield has a circle made around it with pencil, which unfortunately resembles an annular lesion.)

tion of the palms, soles, and flexor surfaces of the forearms. There were a few scattered papules of the abdomen. The top of a papule of the abdomen was abraded and the pulp and serum from beneath, mixed with normal saline, was examined and found to be positive. A firm closed papule of the palm was split and the serum expressed as well as possible. A small amount of material collected on the point of a scalpel was mixed with normal saline and examined by darkfield. It, too, was positive.

No. 9. B. C., a white male, aged 21, was referred with a tentative diagnosis of secondary syphilis. He had seven discrete non-inflammatory lesions of the shaft of the penis, resembling comedones or sebaceous cysts. They were 3 to 4 mm. across and appeared to be almost healed, since there were small scaling areas in the centers. He had an indistinct macular rash of the trunk. There were also a few scattered papules of the trunk and arms. A firm closed papule of the upper right abdominal quadrant was chosen for darkfield examination. The surface was abraded and examination of the exuding serum was done; it was positive. The serologic test proved to be positive.

No. 10. L. W., a colored female, aged 21, was solicited as a marital contact of a patient with secondary syphilis. When she was first seen she had a fissure between the vagina and anus, with a few confluent moist papules about it. There was a scattered papular eruption of the arms and shoulders. Darkfield examinations were as follows:

Date	Source	Number	Result
9-14-42	Vulva	1	Negative
9-15-42	Vulva	4	Negative
9-15-42	Vulva	1	Positive (few seen)
9-15-42	Arm	1	Positive (numerous)

Treatment was begun September 15, 1942.

METHOD

The surface must be abraded vigorously by means of a scalpel or dry gauze. If serum does not exude freely it is necessary to squeeze the area in order to express sufficient material. The treponemes seem not quite as active as those from open lesions, perhaps due to pressure exerted.

SUMMARY

Darkfield examination of either dry or closed secondary lesions of syphilis is a practical procedure, as indicated by the enumeration of the above cases.

REFERENCE

1. Vryonis, G., and Morgan, J. H.: Spirchaete counts in fluid from surface lesions of early human syphilis, Ven. Dis. Inform. 20:343, 1939.

MUMPS

THE RELATIONSHIP BETWEEN SUBMAXILLARY GLAND INVOLVEMENT AND THE INCIDENCE OF ORCHITIS*

A. OPPENHEIM, M. D.
ARCADIA, LA.
AND
HAROLD H. GOLZ, Captain, M. C.
FORT OGLETHORPE, GA.

Mumps is generally regarded as a disease of childhood. Permanent immunity usually results from a single attack, but many practitioners cite examples of second attacks. Mumps can occur at any age, and at any time of the year. Wesselhoeft[1] finds that approximately nine-tenths of city inhabitants above the age of 16 have had mumps, but the percentage in rural communities is much smaller.

IMPORTANCE OF MUMPS DURING WARTIME

The extreme susceptibility of the recruit for contagious disease in general is brought out by Wheelis[2] who showed that by far the highest rates occur during the first two months of service, and that this holds true for all seasons, though less marked in summer than in winter. From a pubilc health and military standpoint, mumps has far more importance than is generally believed. In World War I, one out of every 20 white men, and one out of every six negroes had an attack of this disease. At Camp Wheeler[3] 32 per cent of the soldiers at one time or another during their stay at the camp had mumps. Haven Emerson[4] states that measured by the number of days lost from military service on account of sickness, mumps was the most important disease in the AEF. According to Parran[5] mumps is, next to venereal disease, the most disabling of the acute infections among the recruits. Because of its frequent epidemic proportions, the high rate of hospitalization in the army, and the complications in adults, mumps ranks with many of the more formidable diseases, despite its low mortality.[6]

NATURE OF MUMPS

That the infectious agent of mumps is a filterable virus was established by Johnson

*Published by permission of the surgeon-general of the United States Army.

and Goodpasture[7] in 1934. Today mumps is looked upon by some as a systemic disease, whereas it has usually been considered a local disease of the salivary glands with occasional involvement of other organs "by metastastes." Wesselhoeft[1] believes that Philibert's thesis has stimulated the idea that mumps is to be looked upon as a systemic disease with a variety of manifestations rather than a local disease with complications. Greene and Heeren[8] found 43 nonsalivary manifestations in a study of 100 cases. In a vast majority of cases, the parotid glands are affected; bilaterally in about 70 per cent. Submaxillary glands are frequently involved; the sublinguals occasionally.

ORCHITIS

Other glands in the body may become invaded, especially the gonads in the male. Orchitis rarely occurs before puberty. The incidence is 18 to 20 per cent at and above the age of puberty. While orchitis usually follows parotitis in 10 days, and is more often unilateral than bilateral, evidence seems to indicate that it can precede the appearance of parotitis, occur simultaneously or exist as the sole clinical manifestation.[9] At one time it was thought that the virus was carried by the hand from the mouth to the penis, and that it travelled up the urethra to the testicle. This was disproved by Radin at Camp Wheeler.[3]

Trauma to the testicle during incubation period and during the parotitis has been thought to precipitate testicular involvement. But Wesselhoeft failed to substantiate this.[10] Dukes[12] and Radin[3] have shown that strict bed rest did not reduce the incidence of orchitis. On the other hand, Place[11] reports that only a 5 per cent incidence of orchitis occurred with cases in which thigh bridges with cotton nests and suspensories were used "to protect the testes from trauma and pressure."

Wesselhoeft[9] cites a series of 100 cases in which he administered a preparation of lead. The first 82 did not develop testicular involvement, but in the following 18 cases there were five cases of orchitis, and one of epididymitis; and he abandoned the treatment as futile. Thalhimer[13] asserts that large doses of convalescent serum (40-60 cc.) will be effective in alleviating complications such as orchitis. But Wesselhoeft[9] feels that convalescent serum is of no definite practical value. The treatment of orchitis consists mainly in affording as much relief as possible by means of local applications of heat or cold. Testicular support is comforting. Sedation is usually given. Wesselhoeft concludes that by early operation on the tunica albuginea in severe cases atrophy can be avoided.

RELATIONSHIP OF SUBMAXILLARY GLAND INVOLVEMENT AND ORCHITIS

In reviewing the literature we have not found any direct reference to the relationship of submaxillary involvement in mumps to subsequent orchitis. In cases observed at the Station Hospital, Fort Oglethorpe, Georgia, we noted that patients with parotitis and submaxillary involvement were more prone to develop orchitis than those who had parotitis only. In an effort to establish the validity of this observation, we made careful studies of the following 100 consecutive patients. Our patients were enrollees of the CCC, 4th Corps Area, ranging in age from 17 to 22, and were all from rural communities. The majority of those developing submaxillary gland involvement did so after the parotitis. The submaxillary enlargement usually preceded the orchitis by one or two days, and frequently outlasted it.

Table 1 indicates that 41 per cent of the series had submaxillary involvement.

TABLE 1
INVOLVEMENT IN 100 CASES

	Bilateral Parotitis		Unilateral Parotitis
Parotitis only	37		17
Parotitis with submaxillary only	13		10
Parotitis with submaxillary and orchitis	14		2
Parotitis with orchitis only	4		1
Parotitis with submaxillary and epididymitis	0		1
Submaxillary only—no parotitis		(1)	
Total	68	(1)	31

Radin[3] had parotid gland involvement in 4,243 cases with submaxillaries in 440 or about 10 per cent. Wesselhoeft[9] states that

he had submaxillary involvement in about 50 per cent of his cases.

Table 2 indicates that 51.85 per cent of the total bilateral parotid cases with submaxillary involvement developed orchitis.

TABLE 2

INCIDENCE OF ORCHITIS

Total orchitis with bilateral parotitis	18
Total bilateral parotitis with submaxillary involvement	27
Number of above developing orchitis	14

Table 3 indicates that 15.4 per cent of the total unilateral parotid cases with submaxillary involvement developed orchitis. While the above tables indicate the importance of submaxillary involvement to orchitis, its relative importance is much greater in bilateral mumps.

TABLE 3

INCIDENCE OF ORCHITIS

Total orchitis with unilateral parotitis	3
Total unilateral parotitis with submaxillary involvement	13
Number of above developing orchitis	2

SUMMARY

1. Out of the 41 cases with submaxillary involvement (with unilateral or bilateral parotitis) 16 developed orchitis—39 per cent.

Of the 59 cases without submaxillary involvement only five developed orchitis—8.5 per cent.

2. We found the incidence of orchitis to be four and one-half times as great in the group that suffered from submaxillary involvement as in the non-submaxillary group.

3. Out of the 21 cases of orchitis in our series of 100 cases, 16 had submaxillary involvement—76.2 per cent.

4. We found that about three out of every four patients with orchitis had submaxillary involvement as well.

CONCLUSION

From our observation, while the number of cases is not statistically significant, we feel that there exists a relationship between the submaxillary involvement in mumps and the development of orchitis.

REFERENCES

1. Wesselhoeft, C.: Mumps, Medical Progress, New England J. M., 226:530, 1942.

2. Wheelis, J. M., Jr.: A time study of morbidity and mortality in the U. S. Navy, Am. J. Pub. Health, 28:1291, 1938.

3. Radin, M. J.: The epidemic of mumps at Camp Wheeler, October, 1917—March, 1918, Arch. Int. Med., 22:354, 1918.

4. Emerson, Haven: Military Surgeon, 48:642, 1921.

5. Parran, T.: Health and medical preparedness, J. A. M. A., 15:49, 1940.

6. Gordon, J. E., and Heeren, R. H.: The epidemiology of mumps, Am. J. Med. Sci., 200:412, 1940.

7. Johnson, C. D., and Goodpasture, E. W.: An investigation of the etiology of mumps, J. Exper. Med., 59:1, 1934. The etiology of mumps, Am. J. Hygiene, 21:46, 1935.

8. Greene, J. A., and Heeren, R. H.: Mumps, J. Lab. & Clin. Med., 23:129, 1937.

9. Wesselhoeft, C.: Mumps, Oxford Medicine, 5:489.

10. Wesselhoeft, C.: Orchitis in mumps, Boston M. & S. J., 183:425, 458, 491, 520, 1920.

11. Place, E. H.: Certain aspects of diagnosis and treatment of contagious diseases, Bull. New England M. Center, 3:217, 1941.

12. Dukes, C.: The orchitis of mumps, Lancet, 1:26, 1900.

13. Thalhimer, W.: The prophylactic and therapeutic use of convalescent serum: scarlet fever, mumps and chicken pox, J. Pediatrics, 14:257, 1939.

MENINGITIS CAUSED BY THE HIGHER FUNGI

JOHN E. SKOGLAND, M. D.†

NEW ORLEANS

Although meningitis caused by the higher fungi is comparatively rare, the condition does present some important considerations both to the clinician and the medical bacteriologist. Of the numerous fungi that have been described only four warrant mention in a survey covering infections of the central nervous system. These are Actinomyces, blastomyces, torula and coccidioides.

ACTINOMYCOTIC MENINGITIS

Actinomycosis is mainly a disease of cattle, although it does occur in other animals and in humans. The primary lesion in man is frequently in the oropharynx or adjacent structures, but may be in the lungs, the intestinal tract or the skin. Apparently humans acquire the disease from contact with infected hay or grain. The causative organism, now generally referred to as Actinomyces, has been described in the past under a great variety of names.

Infection of the central nervous system by Actinomyces almost always occurs fol-

†From the Division of Neuropsychiatry, Louisiana State University School of Medicine, New Orleans, Louisiana.

lowing a primary infection elsewhere in the body. The usual method of spread to the meninges is by direct extension from a primary focus in the soft tissues or bony cavities of the head. This is most often an infection in the jaw, but the original lesion may be in some other portion of the face, in the middle ear, the nasal sinuses, the mouth or throat. The infection usually extends intracranially through the foramina at the base of the skull but at times this extension occurs directly through the bone. Infection of the meninges also may occur secondary to a focus which is not contiguous to the central nervous system. In a great majority of cases falling into the latter category, the source of the infection is intrathoracic, either the lungs or bronchial glands. Occasionally it is located elsewhere; for example, in the intestinal tract or skin; in these instances there may or may not be an intermediate stage of pulmonary involvement. When metastatic in this fashion, infection of the meninges always results from invasion of the blood stream. Other secondary lesions may result also once generalized septicemia has become established. Whether infection of the central nervous system is ever primary, in the sense that it is the sole actinomycotic lesion existing, is a debatable issue; certainly, this is a rare phenomenon, if it occurs at all.

Actinomycotic meningitis is most common in young and middle aged adults but it may develop at any age. The majority of afflicted individuals live in rural districts. That observation is understandable in view of the prevalence of the infection among domestic animals; yet, paradoxically, very few humans who become infected give a history of direct contagion.

The pathologic reaction in actinomycosis of the central nervous system may take several forms.[1] Meningitis is most common. The inflammatory exudate in the subarachnoid space is very thick and usually limited to the base of the brain. The pus has a yellowish tinge. Organization may occur with partial obliteration of the subarachnoid space by fibrous tissue. On microscopic examination, the exudate is found to be composed principally of leukocytes, fibroblasts and fungi. Another pathologic process which may exist, either alone or in conjunction with meningitis, is brain abscess. Abscesses of this type are of variable size; they are usually single but may be multiple. The cerebral substance is their favorite location but, strangely, the hypophysis is involved frequently. Cerebellar involvement is rare. Most actinomycotic abscesses are similar grossly and histologically to other chronic types, but their true nature can be recognized by the presence of the characteristic fungi. Occasionally an encapsulated gelatinous or granulomatous lesion forms, differing from the more common suppurative process.

The symptomatology of actinomycosis of the central nervous system is variable. Many clinical features depend upon the type of pathologic involvement which is, as has been mentioned, irregular. Focal symptoms or signs frequently exist in association with a brain abscess, the nature of these being conditioned by the size and location of the lesion. The commonest manifestations of this sort are hemiparesis with the allied reflex alterations, convulsions and visual field defects. Additional symptoms and signs present in many cases include headache, dizziness, vomiting, papilledema, cervical rigidity, positive Kernig sign, muscular hypertonicity, generalized hyperreflexia, delirium and terminal coma. The foregoing manifestations are not specific and may be encountered in association with brain abscess or meningitis caused by any of the common pyogenic organisms. The following are other features of the clinical picture more characteristic of this infection. Fever is usually mild, seldom going above 101° F. The course of the disease is relatively chronic, almost invariably extending over a period of several months, sometimes persisting for over a year and occasionally up to two or three years. There is surprisingly little systemic response to the infection; almost until the time of death, the patient appears comparatively well, even though profound changes have

been produced in the cerebrospinal fluid. Abnormalities in the cerebrospinal fluid may precede by weeks or months development of the initial clinical symptoms and signs of meningitis. Diagnosis is facilitated by the recognition of other actinomycotic lesions extending outside the central nervous system.

Characteristically, the cerebrospinal fluid is extremely purulent, sometimes being so thick that it will scarcely flow through a lumbar puncture needle. The fluid often coagulates after being removed from the subarachnoid space. Fluid so markedly purulent is seldom found under other circumstances and it is suggestive of actinomycotic meningitis particularly, when minimal clinical findings exist coincidentally. The cells usually number several thousand per cubic millimeter and are mostly polymorphonuclear leukocytes. The total protein content is elevated considerably; the sugar and chloride contents usually are low. When a small quantity of cerebrospinal fluid is spread thinly on a slide, the characteristic yellow tinged "sulphur" granules sometimes may be seen with the naked eye. Stained preparations reveal the granules to have both gram-negative and gram-positive components. The organism may be cultured from the cerebrospinal fluid; generally this can be accomplished best under anaerobic conditions, although occasionally it grows aerobically. Isolation of the fungus from cerebrospinal fluid is not feasible always. Animal inoculation is of little or no value as an aid to diagnosis.

The prognosis in actinomycoctic meningitis is poor; all cases terminate fatally. Therapy is entirely symptomatic, although empirically iodides may be prescribed.

BLASTOMYCOTIC MENINGITIS

Infection with Blastomyces (Oidiomyces) occurs in a cutaneous form, with primary involvement usually on the face or hands, and in a systemic form, where widespread dissemination of the organism occurs following invasion through the respiratory tract. Metastatic lesions, associated almost solely with the systemic form of blastomycosis, may develop in skin, bones, spleen, liver, kidneys, lymph nodes and rarely in the central nervous system. The fungus reaches these organs through the blood stream. Infection of the brain and meninges may be preceded by involvement of bone or soft tissue adjacent to the central nervous system, with subsequent direct spread to the contiguous nervous tissue. In other cases, the intracranial infection results from pyemia per se without that intermediate stage. A primary form of meningeal blastomycosis, with no definite manifestations of systemic invasion, has been described also but very likely this interpretation is misleading.[2,3]

The pathologic changes in the central nervous system are, for the most part, nonspecific and closely resemble those associated with tuberculous meningitis. A moderate amount of exudate forms, especially at the base of the brain. Firm nodules, ranging from a millimeter up to several centimeters in diameter, may be situated over the surface of the brain. These nodules are composed principally of fibrous tissue and their central portions often become necrotic. Lymphocytes, endothelial cells, giant cells and organisms are fairly abundant in such lesions. The tendency to caseation and abscess formation is rather pronounced.

Blastomycotic meningitis is characterized by a course usually lasting several months. The symptomatology is the same as in other forms of chronic meningitis. Focal signs and cranial nerve palsies are present occasionally. Fever may be high and is often of an irregular type. Coma supervenes eventually and the outcome is always fatal. The clinical diagnosis is facilitated by the finding of other manifestations of the disease. The skin lesions, characteristically, are elevated, ulcerated areas; abscesses in subcutaneous tissue also exist. Invasion of bone produces localized destruction. Pulmonary involvement, which is exceedingly common, is revealed by a persistent productive cough.

The cerebrospinal fluid frequently is under increased pressure and cloudy. The

cell count is slightly to moderately increased, seldom exceeding 500 per cubic millimeter. Usually polymorphonuclear leukocytes predominate, but this is not true invariably. The total protein may be elevated and the sugar and chloride content reduced. The organism may be seen on direct examination of unstained cerebrospinal fluid, appearing in the form of a round, refractile cell which has a thick wall. Its size is variable but usually the cell diameter is between 10 and 20 microns. Budding forms may be found. In cultures these cells grow in the form of a white mold. The organism may be grown at room temperature on glucose agar.

There is no satisfactory therapy for any form of blastomycosis. Roentgen therapy, excision and iodides have been used in treating localized lesions, but it is doubtful that these measures have any beneficial effects after meningeal invasion has resulted.

TORULA MENINGITIS

Fungi of the genus Torula are widely distributed in nature. They have been cultivated from sources such as the earth, insects, insect nests, plants, trees, fruits, milk and butter. Many varieties have been described. The species causing human infection is known as *Torula histolytica*. This organism possesses definite affinity for nervous tissue and generally the outstanding clinical features of the infection are referable to involvement of the central nervous system. The only other issue for which the organism has any strong predilection is lungs. Occasionally involvement of lymph nodes, intestine, liver, spleen or adrenals is manifest. Skin lesions are present very rarely.

This infection is most common in adults but occasionally it develops during childhood. Males are afflicted more often than females. Apparently occupation plays no significant role. The respiratory tract is the usual portal of entry, when the organism invades the human body. Dissemination then takes place through the blood stream or lymphatics with involvement of the central nervous system resulting almost invariably, and in at least half the cases, involvement of other organs occurs also.

The pathologic changes in the central nervous system are variable. Freeman has classified these into three types—meningeal, perivascular and embolic.[4] Nearly half the cases are purely meningeal. This reaction bears close resemblance to that occurring in tuberculous infections. Microscopically, the meninges show a lymphocytic infiltration, localized fibrosis, giant cell formation and occasionally caseation. Organisms may be abundant. Perivascular changes of a greater or lesser degree are present in most cases. Where superficial vessels are involved, it is likely that the perivascular spaces have been invaded by direct extension from the subarachnoid space. Similar changes about deep vessels are related to embolic foci. Granulomas and cysts are other lesions of embolic origin. These occur in the basal ganglia, the mid-brain and the cortex. The cysts, which typically are multiocular, contain a gelatinous fluid and numerous organisms. They may be multiple and are sometimes very large. They rupture occasionally. In some instances microscopic studies of brain tissue have revealed an increase of neuroglia, degenerative changes in ganglion cells and degeneration of myelin sheaths.

The symptomatology of torulosis varies with the nature and extent of the pathologic changes. The predominant clinical features are referable almost always to invasion of the central nervous system. Diverse symptoms are caused by foci in other organs. Usually the initial manifestation is intermittent frontal headache; this gradually becomes more severe and continuous. Occasionally the disease begins abruptly with an excruciating persistent headache. Anorexia, vomiting, vertigo, backache, stiffness of the neck and positive Kernig sign are additional early features. Irritability, insomnia and other mild changes in personality are fairly common; ultimately, a frank organic psychosis may evolve. Cranial nerve disturbances occur irregularly. Papilledema, diminished vision, strabismus, diplopia, ptosis, and deafness have been re-

ported. Reflex alterations are found often. Gross focal manifestations, such as hemiparesis or aphasia, exist at times. As the disease advances, emaciation and profound debility appear. The course is steadily progressive over a period which generally ranges between a few weeks and six months but which may extend for a year or longer. Remissions occur rarely. There is no acute systemic reaction to the infection. The temperature and pulse remain normal or nearly normal. A mild leukocytosis or anemia may develop. Coma supervenes terminally and the infection always proves fatal.

Usually the cerebrospinal fluid is under increased pressure, though this finding is not constant. The fluid may appear clear, turbid or, rarely, xanthochromic. Commonly it contains several hundred cells per cubic millimeter but the cell count may range from normal up to approximately one thousand cells per cubic millimeter. These cells are chiefly or entirely lymphocytes. Both the sugar and chloride levels may be reduced. The total protein content is often elevated. The collodial gold curve is abnormal but shows no typical alteration. Yeast-like cells may be visible on direct examination of the cerebrospinal fluid; these are round or oval and range from a few microns up to about 50 microns in diameter. Many have a double wall and some show budding. Organisms can be identified in unstained preparations. They may be seen in the counting chamber among the white blood cells, and at times are more numerous than the latter. They are often present in great numbers in the sediment following centrifugalization. Although the fungi are gram-positive, it is difficult to recognize them in stained smears. Torulae grow on ordinary culture mediums but growth is most rapid on Sabouraud's medium. Cultures of the cerebrospinal fluid are usually positive. Diagnosis is aided by the inoculation of cerebrospinal fluid, or substance from a culture, intraperitoneally in white mice. After mice succumb to the infection, fungi may be isolated easily from various organs.

There is no specific treatment for this disease. However, the common symptomatic measures may produce temporary relief.

COCCIDIOIDAL MENINGITIS

Coccidioidal granuloma is an endemic disease having a limited geographic distribution. It occurs principally in the San Joaquin Valley, California, but a few cases have been reported also from nearby regions.

The fungus causing the infection is *Coccidioides immitis*. It gains entrance to the body through either the lungs or the skin, and subsequently is carried by the blood stream to other organs. Infection of the central nervous system occurring in this manner may exist as the only metastatic lesion or along with various others. Approximately 25 per cent of patients with coccidioidal granuloma develop infection of the meninges.[5] The disease occurs usually in adults and is more common in males than females.

The pathologic findings to some extent vary with the duration of meningeal involvement. An exudate accumulates in the subarachnoid space and eventually it becomes organized. This inflammatory reaction is most pronounced at the base of the brain. Frequently the drainage of cerebrospinal fluid is interfered with and internal hydrocephalus results. In chronic forms, granulomatous lesions are common. Small nodules are found over the dorsolateral surfaces of the brain. Larger lesions tend to occur in the basilar cisterns and about the spinal cord. Histologically, the meningeal reaction resembles that associated with a tuberculous infection, except for the presence of the offending organism. Direct involvement of the neural parenchyma is rare.

The symptoms of coccidioidal meningitis are non-specific. They are similar to those observed in other chronic meningitides, with evidences of internal hydrocephalus often superimposed. The outstanding manifestations include headache, nausea, vomiting, cervical rigidity, occasionally papilledema and in late stages stupor. Sometimes, though not always, coccidioides infection of other body organs may be evident. The

skin lesions are variable but multiple subcutaneous abscesses and chronic ulcers are seen most regularly. Pulmonic involvement produces symptoms and roentgenographic changes suggestive of pulmonary tuberculosis. The systemic reaction to the infection is characterized by malaise, cachexia, generalized weakness, slight fever and a moderate leukocytosis. Only rarely is the organism found in the blood. The duration of the disease, after the onset of meningeal symptoms, varies from a few days up to one year and in most cases extends over a period of several months. Diagnosis is aided sometimes by roentgenographic demonstration of erosion of the inner table of the skull or involvement of vertebrae.

Changes in the cerebrospinal fluid closely resemble those occurring in tuberculous meningitis. Generally the fluid is under increased pressure. It may appear clear, turbid or xanthochromic. The cell count averages several hundred per cubic millimeter but may be as high as 2000 per cubic millimeter. Lymphocytes predominate. The total protein may be elevated, the sugar and chloride values diminished. Usually the colloidal gold curve is of the first zone type. The organism may be isolated from the cerebrospinal fluid, although this is not always possible. The fungus is spherical, has a double contoured refractile wall and ranges between 5 and 50 microns in diameter. In the process of reproduction, a number of smaller cells form from the protoplasm; after rupture of the cell wall, these are liberated and develop into adult forms. Budding is not seen. The organism may be identified on microscopic examination of unstained preparations. In stained preparations it is gram-negative. In artificial cultures, it appears as a white mold. Sabouraud's medium is very suitable for its growth. Confirmation of the diagnosis may be obtained by animal inoculation; four to six weeks after injection, intraperitoneally, the fungus will kill a guinea-pig and lesions revealed at necropsy are similar, apart from the organism, to those associated with tuberculosis.

Coccidioidal meningitis always terminates fatally. Various drugs have been used in the treatment of coccidioides but none has given consistently satisfactory results. Symptomatic therapy is the same as for other forms of meningitis. When localized compression of the spinal cord exists, laminectomy is justified sometimes in an attempt to remove granulomatous tissue.[6]

SUMMARY

The neurologic aspects of fungus infections have been emphasized in the foregoing review.

The syndromes discussed are characterized clinically by the occurrence of a chronic meningitis, associated at times with focal symptoms and signs. The diagnosis can be established positively only by identification of the parasite.

REFERENCES

1. Friedman, E. D., and Levy, H. H.: Actinomycotic infection of the central nervous system: report of a case and review of the literature, Internat. Clin., 2:36, 1937.

2. Wilhelmj, C. H.: The primary meningeal form of systemic blastomycosis, Am. J. Med. Sci., 169:712, 1925.

3. McBryde, C. M., and Thompson, E. I.: Meningitis and dermatitis caused by a new variety of blastomycete (endomycete), Arch. Derm. & Syph., 27:49, 1933.

4. Freeman, W.: Torula infection of the central nervous system, J. f. Psychol. u. Neurol., 43:236, 1931.

5. Abbott, K. H., and Cutler, O. I.: Chronic coccidioidal meningitis, Arch. Path., 21:320, 1936.

6. Rand, C. W.: Coccidioidal granuloma, Arch. Neurol. & Psychiat., 23:502, 1930.

THE CRUVEILHIER-BAUMGARTEN SYNDROME

FRANCIS E. BRUNO, M. D.*

NEW ORLEANS

Recently a case of cirrhosis of the liver was observed which clinically manifested the signs of portal hypertension and which also showed evidences of collateral circulation as manifested by the presence of a roaring murmur and thrill over the area of the ensiform process. Such a syndrome has attracted but scant attention in the American literature, since most of the cases have been reported in the German, French, English and Italian journals. However, a short

*From the Department of Medicine, School of Medicine, Tulane University of Louisiana and Charity Hospital of Louisiana, New Orleans, La.

while ago an excellent review of the literature on this case syndrome revealed 52 cases and contained two additional case reports.

Cruveilhier[1a] described a patient who had originally been observed by Pegot in 1833. This patient showed dilated abdominal veins, a caput medusae, and a loud murmur in the umbilical area. Postmortem examination disclosed a large spleen, a grossly normal liver, and a widely patent umbilical vein. Baumgarten[1b] reported a case with distended abdominal veins, ascites, splenomegaly, anemia, and leukopenia. No epigastric murmur was heard. Autopsy revealed an atropic liver, splenomegaly and a widely patent umbilical vein. Since that time numerous authors have reported similar cases which presented an epigastric murmur frequently associated with a thrill and concomitant signs of portal hypertension. Necropsy in the majority of cases has revealed cirrhosis of the liver with excessive portoumbilical circulation. European authors have attached to this combination of findings the term "Cruveilhier-Baumgarten syndrome." Green[2] states that this term may be applied to patients having the clinical picture of portal hypertension featured by evidence of an excessive umbilical circulation and the presence of a loud abdominal murmur and thrill.

Armstrong[1] and his associates divide these cases into several groups which have more pathologic than clinical significance. They separate the cases which at postmortem show patency of the umbilical vein, atrophy of the liver with little or no cirrhosis and splenomegaly into what they term "Cruveilhier-Baumgarten disease." All other cases of portal hypertension from disease of the liver or portal system with patent umbilical veins or patent paraumbilical veins, or with patency of other collateral veins in the area are classified as "Cruveilhier-Baumgarten syndrome."

Frequently the murmur and thrills due to the collateral circulation in this syndrome has been mistaken for evidence of organic heart disease. Indeed one author[3] has stated that this condition should be considered on a differential diagnosis of congenital heart disease. I wish to report a case in which at first the possibility of an arteriovenous fistula was strongly entertained.

CASE REPORT

T. D., a 48 year old, white, married lumber worker was admitted to the Charity Hospital Medical Service (Tulane Unit) October 25, 1941. The patient's reason for coming to the hospital was that he had been advised to do so by his attending physician because of "liver trouble."

Seven years prior to admission the patient experienced an attack of jaundice, which was asymptomatic except for a little fever. In the following five years he had four such attacks. There was no history of colic, melena, abdominal pain or clay colored stools. Prior to the first episode the patient had drunk one or two drinks of liquor daily, but upon the advice of his physician he had stopped the use of alcohol absolutely.

The patient then felt perfectly well until September 1, 1941, when he developed a slight fever. The fever abated after he had taken quinine daily for seven days. On October 22, 1941, the patient was suddenly seized with a severe epigastric pain which radiated to both axillae. This pain was not associated with vomiting, diarrhea, or melena, nor was it colicky in character. After about three hours the acute pain disappeared, leaving a residual soreness which was present on admission three days later. During the acute attack the local physician noticed that the abdomen was distended and that edema of the ankles, legs and scrotum were present.

The only positive findings in the system inventory was deafness of many years' duration, ever since past employment in a boiler factory. The previous history and family history were not significant.

Physical examination showed a middle-aged white male who was not acutely ill. The abdomen was protuberant, but there was no wasting of the upper part of the body. The skin was warm and elastic, several spider angiomata were apparent over the thorax, just beneath the right sternoclavicular joint, and one was present on the left shoulder. These consisted of a red central body from which radiated many legs, and which blanched on pressure over the center. The angioma on the left shoulder pulsated synchronously with the patient's pulse. There was no visible evidence of collateral circulation or varicosities over the abdomen and thorax. A cleft in the sternum could not be palpated. There was impairment of hearing. The periphery of the sclerae was slightly icteric. Many teeth were carious. Examination of the heart and lungs showed no abnormalities.

The abdomen was symmetrically distended. Shifting dullness and fluid wave were not demonstrable. The liver and spleen were not felt. There was edema of the scrotum and of the prepuce.

The ankles and legs presented slight pitting edema. Rectal examination revealed external hemorrhoids.

Laboratory data: The urine was essentially normal, except for a slight trace of bile. The icteric index was 18 units. The total protein was 6.16 mg. The blood cholesterol was 83 mg. per cent. The hemoglobin was 60 per cent; erythrocytes 3,500,000; leukocytes 2,500; with 56 per cent neutrophiles, 36 per cent lymphocytes, and 8 per cent monocytes. The blood serology was negative.

The Takata Ara was positive in tubes 1, 2, and 3. The cephalin flocculation test was 3+ positive. Intravenous hippuric acid excretion was only 0.18 gm.

The roentgenogram of the chest showed nothing of note, except a cervical rib on the right side. Infra-red photography of the abdomen did not disclose any distended abdominal veins. Clinical diagnosis: Cirrhosis of the liver.

Special examination: On palpation of the chest a fine thrill was felt over the xiphoid process. On auscultation a loud roaring murmur was heard.

The thrill and murmur were not invariably present. The character of the murmur was like a strong wind but it was not constant, dying down at times and reappearing. Pressure with the flat end of a pencil slightly to the left of the xiphoid process caused complete disappearance of both murmur and thrill. When the pressure was terminated, both thrill and murmur reappeared. Change of position from one side to the other, or from one side to the dorsum, did not appear to alter the bruit. At times the murmur and thrill were manifest in the erect position, and at other times they were absent.

The murmur had a marked respiratory rhythm. The greatest intensity of the murmur developed during inspiration but the increase in intensity began during the latter part of expiration. During a number of periods of study the peaks of intensity due to respiration varied greatly since there was a gradual waxing and waning through a period of 10 to 40 respiratory cycles.

At times there was a definite accentuation of the murmur due to the heart beat except for a few special circumstances. It became completely continuous without any respiratory effect following the drinking of warm broth, the injection of histamine and one occasion during a fasting state. This last observation was made during a period of study after the patient had suffered from an acute cellulitis of the leg for which he was treated with sulfathiazole. When the patient stopped breathing, the murmur became steady in its intensity and frequently would show diminution after a period of 20 to 30 seconds. Abdominal pressure intensified the murmur.

The intensity of the murmur and the response to various procedures was registered graphically by Dr. R. H. Turner in his laboratory.

COMMENT

The striking conversion of the bruit from a relatively intermittent murmur at first into a continuous loud roar by the administration of histamine or the drinking of warm broth is evidence that one of the factors governing the intensity of the murmur is the rate of blood flow through the splanchnic system.

DISCUSSION

There has been considerable speculation concerning the exact nature of the disturbances giving rise to such circulatory manifestations. Various theoretical anatomic variations which might set the venous column of blood vibrating with the resultant production of the thrills and bruits characteristic of the Cruveilhier-Baumgarten syndrome have been put forward as an explanation. Both Thayer[4] and Lutembacher[5] have discussed in their articles the possible causes of the venous murmur and thrill occurring in cirrhosis.

One author stated that the cause of the signs was due to the transmission of a murmur produced by venous eddies set up at the entrance of the vena cava into the right auricle. Other concepts were that pressure[6] traction, or distortion of the vena cava might be the cause of venous disturbances. Dilated coronary veins, esophageal varices, arteriovenous anastomoses[7] and anemia have all been incriminated as the causative factor of this circulatory disorder. Thayer pointed out the features which invalidated these assumptions. He felt that a constricted channel passing to a dilated vessel deep in the abdomen would not be manifested by murmurs and a thrill superficially. It is indeed hard to conceive that palpable thrills or loud murmurs could arise from such disturbances. Neither could light compression cause thrills and murmurs to disappear if deep fluid eddies were the cause of such murmurs or thrills.

In the majority of cases presenting a palpable thrill and continuous murmur, postmortem examination revealed a peculiar configuration of the porto-umbilical venous network. The abnormalities were not constant in their architecture but any of those

described can readily explain the phenomenon. In many of the reported cases presenting the features diagnostic of the syndrome, which have come to necropsy, persistent umbilical veins or paraumbilical veins were found to be patent.[5] Whether these paths of collateral circulation were congenitally functional or whether their patency was resumed secondary to portal obstruction can only be conjectured. In the majority of cases the umbilical vein or paraumbilical veins derived a profuse collateral delivery from anastomoses with the thoraco-abdominal veins. In some instances the presence of fibrous bands constricting the accessory circulation was an additional factor in circulatory obstruction. An excellent discussion of these venous paths, is given in the article by Armstrong[1] et al.

An anatomic feature which was found to be prominent in many cases, and which I feel is clinically significant, has been found at postmortem in many of the cases. Gwyn,[3] in an excellent report of a patient in whom the murmur disappeared on pressure, pointed out a structural arrangement which had been noted before. In his patient the vein appeared to run up the sternum and perforated a small fissure at the xiphosternal juncture. Immediately beneath the sternum and xiphoid this vein could be seen to enter a large venous sinus 1 to 4 cm. which was situated in the areolar tissue just under the bone. Into the sinus and coming directly through the diaphragm ran another large vein, and this vein in turn could be seen to come from the suspensory ligament and to be connected with moderately distended umbilical veins. From the upper point of this sinus there was a wide open venous connection with the much dilated internal mammary vein. Palpation of the sternum revealed the cleft in this case. Gwyn concluded that defects in ossification of the sternum producing such clefts are a not uncommon anomaly. The collateral venous trunks arising in the course of cirrhosis may easily pass through such routes to sinuses under the sternum. Fluid eddies set up by blood passing from a constricted channel to a dilated sinus may give rise to loud murmurs and thrills in such cases.

Henry[8] described an instance in which a loud roaring murmur was heard over the ensiform which disappeared on light pressure. Under the ensiform cartilage and extending to the articulation of the sternum, there was found a venous sinus about 7 mm. in diameter. The anterior wall of the sinus could be seen through an opening in the ensiform. Proceeding from the opening in the ensiform were several superficial veins probably in communication with the sinus.

Lutembacher[5] reported a somewhat similar case in which chondrofibrous tunnels existed in the xiphoid process. There was a venous plexus deeply situated under the right rectus which communicated with the deep epigastric and the internal mammary vein.

It is felt that this same situation obtained in the case which I have reported here. The reason for believing that there was a cleft in the sternum is that the vein could be traced up to the xiphoid, and pressure at any point along the course of the vein caused both murmur and thrill to disappear. Localized pressure over the xiphoid with the tip of a pencil obliterated both murmur and thrill. Unfortunately the sternum was not x-rayed for any osteochondral defect.

It is suggested that in patients presenting the signs of portal obstruction associated with a murmur or thrill or both, which may be made to disappear by compression, that collateral circulation dependent on defects in the sternum may be present. Palpation or radiologic examination may reveal such defects.

The most recent review of the literature on the Curveilhier-Baumgarten syndrome listed 54 cases of this interesting condition. Search of the literature reveals an additional 11 cases previously recorded in which the clinical picture was consistent with the Cruveilhier-Baumgarten syndrome. Gywn,[3] Bates,[9] Hall,[10] Wilson[11] and Mirvish[12] have each reported a single case. Kenawy[13] states that he has observed six cases associated with bilharzial cirrhosis of the liver. This with the case described here adds 12 cases,

so that a total of at least 66 cases of the Cruveilhier-Baumgarten syndrome have been reported to this date.

SUMMARY

A case of cirrhosis of the liver which fulfills the requirements of Cruveilhier-Baumgarten syndrome, namely clinical portal hypertension featured by a loud epigastric murmur and thrill, is reported.

Attention is called to a fact which has hitherto not been stressed, that in cases of this syndrome in which the murmur or thrill may be altered by compression, there may coexist an osteochondral defect of the sternum.

REFERENCES

1. Armstrong, E. L., Adams, W. L., Tragemenn, L. J., Townsend, E. W.: The Cruveilhier-Baumgarten syndrome. A review of the literature and report of two additional cases, Ann. Int. Med., 16:113, 1942.

1a. Cruveilhier, J.: Cited by Armstrong

1b. Von Baumgarten, P.: Cited by Armstrong

2. Greene, C. H.: Liver and biliary tract, Arch. Int. Med., 60:691, 1942.

3. Gwyn, N. B.: On some venous murmurs found in hepatic cirrhosis and their confusion with murmurs of congenital heart diseases, Am. J. Med. Sci., 180:525, 1930.

4. Thayer, W. S.: On the presence of a venous hum in the epigastrium in cirrhosis of the liver, Am. J. Med. Sci., 141:313, 1911.

5. Lutembacher, R.: Bruits de souffle vinlux et cirrhose de luevee, Press med. Paris, 44:847, 1936.

6. Rolleston, H. D., and McNee, J. W.: Diseases of the Liver, MacMillan Company, London, p. 267, 1929.

7. Wollager, E. E., Keith, N. M.: Epigastric thrill and murmurs in a case of cirrhosis of the liver with general vascular signs of arteriovenous fistula, Proc. Staff. Meet. Mayo Clinic, 13:33, 1938.

8. Henry, N. P.: Report of a case of loud venous hum heard over the xiphoid cartilage in cirrhosis of the liver, quoted by Armstrong et al.

9. Bates, J. L.: Continuous venous hum in cirrhosis of the liver, Lancet 1:1108, 1937.

10. Hall, A. J.: Continuous venous hum in cirrhosis of the liver, Lancet 1:1202, 1937.

11. Wilson, L. A.: Continuous venous hum in cirrhosis of the liver, Lancet 2:745, 1946.

12. Mirvish, J.: Continuous venous hum in cirrhosis of the liver, Lancet 2:142, 1937.

13. Kenawy, M. R.: Continuous hum in cirrhosis of the liver, Lancet 1:281, 1937.

NEW ORLEANS
Medical and Surgical Journal

Established 1844

Published by the Louisiana State Medical Society under the jurisdiction of the following named Journal Committee:

Emmett L. Irwin, M. D., Ex officio
For one year: Leon J. Menville, M. D., Chairman.
D. I. Hirsch, M. D.
For two years: Lucien A. LeDoux, M. D., Vice-chairman
H. W. Kostmayer, M. D. Secretary
For three years: James K. Howles, M. D.

EDITORIAL STAFF

John H. Musser, M. D.....*Editor-in-Chief*
Willard R. Wirth, M. D.....*Editor*
Edwin L. Zander, M. D.....*Associate Editor*

COLLABORATORS—COUNCILORS

H. E. Bernadas, M. D.
C. Grenes Cole, M. D.
Guy R. Jones, M. D.
M. D. Hargrove, M. D.
John G. Snelling, M. D.
Rhett McMahon, M. D.
C. A. Martin, M. D.
W. F. Couvillion, M. D.

Paul T. Talbot, M. D.....*General Manager*
1430 Tulane Avenue

SUBSCRIPTION TERMS: $3.00 per year in advance, postage paid, for the United States; $3.50 per year for all foreign countries belonging to the Postal Union.

News material for publication should be received not later than the eighteenth of the month preceding publication. Orders for reprints must be sent in duplicate when returning galley proof.

Manuscripts should be addressed to the Editor, 1430 Tulane Avenue, New Orleans, La.

THE JOURNAL *does not hold itself responsible for statements made by any contributor.*

THE RATIONING OF GASOLINE

Elsewhere in this issue of the Journal is published a letter that was sent out by one of the county medical societies of Texas to its constituent members. This has to do with the use of the physician's automobile now that gasoline rationing is in effect. It will be well worth while if every doctor were to read these words of advice issued by the Tarrant County Medical Society.

The physician now is in a rather peculiar situation on account of gas rationing. Doctors can get "C" cards because an automobile is necessary for them in the practice of their profession. The doctor will be watched extremely carefully. People are always likely to criticize when they feel that they are not being treated entirely justly. There is a good deal of feeling against gasoline rationing, more particularly by selfish individuals who are not willing to give up and make sacrifices for the war effort. If these people see the doctor using his car and driving it on non-professional trips it is going to cause gossip, possibly ill feeling. The physician must be extremely punctilious and very careful about using the car for social and pleasure trips. The same restriction applies to his family.

Already in the East, where rationing has been in effect for some months, some doctors are being censured for non-professional use of their automobiles. Even in the daily press, comment has been made about doctors' cars being present at racing meetings and sporting events. Our profession is an honored one, the physician is always looked upon as being a respected and honored member of the community. It is decidedly up to us to see that nothing is done in this car rationing plan to lower the respect and honor that we have in the community.

NUTRITION IN THE AGED

The feeding of the old person is frequently a difficult undertaking. The old individual has cultivated a taste for certain foods during the course of his life and a distaste for others. The latter group is often composed of the foods which are the most nourishing and which are highest in their vitamin and mineral content. Furthermore, the old individual has often a poor appetite which has to be pampered and lastly he is handicapped in many instances by dentures which fit poorly, producing ulcers in the mouth and which, even if no local lesions are brought about, do not permit proper mastication of the coarser dietary constituents. He learns to eat concentrated foods, soft, pappy articles which again do not supply the vitamins and minerals necessary for well being.

As a result of the dietary bad habits of the older individual, it is common to find in these persons evidences of vitamin deficiency. These deficiencies are usually those which are labeled chronic dietary deficiency and are multiple rather than single. The vitamin B complex is the one vitamin which is most likely to be inadequate for the maintenance of good health. These older persons frequently have cracks and fissures in the angles of the mouth, atypical cheilosis; often the tongue is a deep, somewhat fiery red, with well defined fissures. The skin is unusually thickened and pigmented, especially over the bony prominences. They will complain of nervousness, the family recognizes their irritability, diarrheas are common, while the mental expressions are those that often are attributed to old age, such as mental confusion, forgetfulness, depression and anxiety which, it is true, are common in old age but which can be improved materially by an adequate diet. The older woman may have a chronic vaginitis, or proctitis may be present in either sex as result of inadequate diet. The chronic conjunctivitis and lacrimation of old people is said to be due in part to vitamin A deficiency. There is a host of other rather indefinite, somewhat vague objective findings which, in the opinion of most nutritionists, point to inadequacy of the diet.

In a recent survey of two hundred patients, Goldsmith found many of these evidences of dietary inadequacy are present in the older aged group. The arteriosclerotic persons to a large per cent showed the findings of a vitamin lack, particularly in the white race.

The obvious implications are that the older person should observe proper dietetic hygiene, just as the younger adult should eat the sane and sensible foods, or as the competent physician insists that his baby and children patients should be fed. As a matter of fact it is more necessary for the older person to take in a greater amount of vitamins and minerals than does the younger adult because in the aged patient storage may be poor, utilization imperfect and excretion rapid.

VIRUS PNEUMONIA

A recent review* brings out the pertinent and known facts regarding a most interesting condition which has been widely observed throughout the country. Although the general concept is that this is a relatively new disease, actually it was probably as long ago as 1872 that this peculiar type of pneumonia was recognized.

A variety of names have been given to this disease, commonest of which is a virus pneumonia, but it has also been called an acute pneumonitis, acute interstitial pneumonitis, atypical pneumonia with leukopenia, bronchial pneumonia of unknown etiology and many other terms. In order to classify accurately this disease, the Pneumonia Commission of the Board for the Investigation and Control of Influenza and Other Epidemic Diseases in the United States Army has designated the disorder as "primarily atypical pneumonia, etiology unknown."

For the purposes of this discussion the name virus pneumonia will be employed although investigators have been unable to reproduce the disease with any of the unknown viruses, or is it known definitely that it is produced by a virus. The ordinary laboratory studies have not identified the causative factor, although many organisms have been incriminated. Elaborate investigations of a laboratory nature have failed to show that the disease is due to any one entity. These studies include intensive use of animal inoculation, with practically negative results.

Virus pneumonia is essentially a disease of the younger portions of the population. Most of the cases are seen in the age group from which the Army is drawn and while the young adults have been the ones most frequently attacked, the age limits run from four to eighty years. Sex distribution is equal between the two sexes. It might be noted here that apparently the disease is not extremely infectious, particularly when

*Dingle, J. H., and Finland, M.: Medical progress: Virus pneumonias: II. Primary atypical pneumonias of unknown etiology, New England J. M., 227:378, 1942.

a patient does not have a cough. However, the doctors and nurses and soldiers living in barracks have been the persons most frequently attacked so that very definitely there must be contact spread of the disorder.

Virus pneumonia occurs with various degrees of severity. In most instances it is associated with fever, cough, headache, chilliness and sweats. Often sore throat is present but lasts for only a day or two. Symptoms in mild cases naturally are not particularly severe but in the more severe cases the symptoms are exaggerated, prostration is often extreme and the temperature may persist for several weeks. Convalescence is prolonged in the more severe cases but death rarely occurs, consequently the pathologic studies are at the best quite skimpy.

One of the most interesting aspects of virus pneumonia is the almost complete absence of physical signs by common methods of examination. In the ordinary case the respirations are not increased, there is no change in percussion dulness or in the character of the breath sounds. Such pulmonary signs as are noted are confined to the presence of subcrepitant rales, generally only brought out by coughing. On the other hand, the x-ray signs are definite and distinct. One of the most remarkable features of the disease is to observe the often extensive lesions in the lungs disclosed by the roentgenogram while the physical signs are virtually absent. The x-ray evidences of the disease may persist for many weeks. Incidentally as evidences of the mildness of the disease it might be stated that in one of the Army camps in this country 100 consecutive chest roentgenograms were made of soldiers who had had a mild cough or "grip" which incapacitated them only for a few days to those who were more severely ill and required hospitalization for several weeks, in practically all of these instances the x-ray evidence showed the lung mottling which is so characteristic of this type of pneumonia. Likewise it should be observed that probably the disease is much more likely to be discovered nowadays than it was in the past with the almost universal use of x-ray in the presence of pulmonary symptoms. Undoubtedly in bye-gone days many of these cases would never have been recognized, possibly the great majority of them.

This is one of the diseases associated with leukopenia and most of such diseases are not helped by the sulfa drugs. The various sulfonamides have been employed without any marked effect on the course of the disease. As a matter of fact it would probably be better not to make use of these drugs because they may possibly cause rather severe reactions and they may furthermore increase the symptomatic discomfort of the patient. The treatment then is essentially the combatting of symptoms; opiates and analgesics to alleviate the pains and aches, expectorants if needed and once in a while oxygen. It probably would be the part of wisdom to isolate the patient.

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

January 4. Orleans Parish Medical Society, Board of Directors, 8 p. m.

January 5. Eye, Ear, Nose and Throat Staff, 8 p. m.

January 6. Clinico-pathologic Conference, Charity Hospital Morgue Amphitheater, 1:30 p. m.
Mercy Hospital Staff, 8 p. m.
Clinico-pathologic Conference, Marine Hospital, 7:30 p. m.

January 7. Clinico-pathologic Conference, Touro Infirmary, 11:15 a. m. to 12:15 p. m.
Baptist Hospital, Executive Staff, 8 p. m.

January 11. Orleans Parish Medical Society, Installation of Officers, 7:30 p. m.

January 13. Clinico-pathologic Conference, Charity Hospital Morgue Amphitheater, 1:30 p. m.
Touro Infirmary Staff, 8 p. m.
Women's Auxiliary, Orleans Parish Medical Society, Orleans Club, 3 p. m.

January 14. New Orleans Hospital Council.

January 15.	I. C. R. R. Hospital Staff, 12:30 p. m.
January 18.	Hotel Dieu Staff, 8 p. m.
	Clinico-pathologic Conference, Baptist Hospital, 8 p. m.
January 19.	Charity Hospital Medical Staff, 8 p. m.
	Clinico-pathologic Conference, Charity Hospital Morgue Amphitheater, 1:30 p. m.
	Charity Hospital Surgical Staff, 8 p. m.
	Clinico-pathologic Conference, Hotel Dieu, 8:15 p. m.
January 21.	Clinico-pathologic Conference, Touro Infirmary, 11:15 a. m. to 12:15 p. m.
January 26.	Baptist Hospital Staff, 8 p. m.
January 27.	Clinico-pathologic Conference, Charity Hospital Morgue Amphitheater, 1:30 p. m.
	French Hospital Staff, 8 p. m.
	Catholic Physicians' Guild, 8 p. m.
January 28.	Clinico-pathologic Conference, Touro Infirmary, 11:15 a. m. to 12:15 p. m.
	DePaul Sanitarium Staff, 8 p. m.
January 29.	L. S. U. Faculty Club, 8 p. m.
	New Orleans Hospital Dispensary for Women and Children Staff, 8. p m.

During the month of December the Society held one regular scientific meeting. The program was as follows:

Glomerulonephritis, by Dr. Edgar Hull.

Hypogastric Sympathectomy, by Dr. B. Bernard Weinstein.

Vaginal Ureter-Lithotomy, by Dr. Robert F. Sharp.

The seventeenth Stanford E. Chaille Memorial Oration was held Saturday, December 19. Dr. Roy Biggs Henline, Associate Attending Urologist: Brady Urology Department, New York Hospital; Riverside Hospital; Attending Urologist, New York Foundling Hospital, was the guest speaker. Dr. Henline spoke on Cancer of the Prostate. Dr. E. A. Ficklen gave the memoriam.

At the annual election of the Society held Saturday, December 12, 1942, the following officers of the Orleans Parish Medical Society and delegates to the Louisiana State Medical Society for 1943 were elected:

President-Elect: Dr. Edwin L. Zander.
First Vice-President: Dr. George H. Hauser.
Second Vice-President: Dr. Philip H. Jones, Jr.
Third Vice-President: Dr. Walter Levy.
Secretary: Dr. Daniel J. Murphy.
Treasurer: Dr. A. V. Friedrichs.
Librarian: Dr. Max M. Green.
Additional Members to the Board of Directors: Dr. Paul G. Lacroix, Dr. E. L. Leckert, and Dr. E. J. Richard.

Delegates to the Louisiana State Medical Society (newly elected):

Dr. Gilbert C. Anderson, Dr. Edgar Burns, Dr. Edmund Connely, Dr. A. V. Friedrichs, Dr. Max M. Green, Dr. George H. Hauser, Dr. Paul G. Lacroix, Dr. Waldemar R. Metz, Dr. Robert Sharp, Dr. Edmond Souchon, Dr. H. Ashton Thomas, and Dr. Edwin L. Zander.

EDWIN L. ZANDER, M. D.,
Secretary.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	

AN IMPORTANT NOTICE

It is of the utmost importance that the Procurement and Assignment Service for Physicians, Dentists, and Veterinarians, immediately has the name of any doctor who really is willing to be dislocated for service, either in industry or in overpopulated areas, and who has not been declared essential to his present locality. This is necessary if the medical profession is to be able to meet these needs adequately and promptly. We urgently request that any physician over the age of 45 who wishes to participate in the war effort send in his name to the State Chairman for the Procurement and Assignment Service in his State.

FRANK H. LAHEY, M. D.,
Chairman, Directing Board.

DR. P. S. PELOUZE TO ADDRESS PHYSICIANS OF LOUISIANA

Dr. P. S. Pelouze, who is known throughout the country for his research in gonorrhea, has been given a leave of absence by the University of Pennsylvania, where for many years he has been Associate Professor of Urology, to speak before medical societies throughout the United States on the subject of the medical management of gonorrhea.

Dr. Pelouze will begin his tour of Louisiana in Shreveport on February 1. His planned itinerary is as follows:

February 1-2	Shreveport
February 3	Monroe
February 5	Bogalusa
February 8-10	New Orleans
February 15	Baton Rouge
February 17	Opelousas
February 19	Lafayette
February 22	Lake Charles
February 24	Leesville
February 25	Alexandria

The parish medical societies in most of these areas have already scheduled meetings at which loyal physicians will have the opportunity to hear Dr. Pelouze. Every effort will be made to schedule additional meetings in other parts of the state if a desire to hear Dr. Pelouze is communicated to the Secretary of the State Board of Health or the Secretary of the Louisiana State Medical Society.

Dr. Pelouze, in addition to his extensive research in the field of urology, is a fluent and interesting speaker. He has also written several books dealing with the treatment of gonorrhea and other diseases in the field of urology.

BI-PARISH

The Bi-Parish Medical Society met for its annual meeting December 2, in the Rist Hotel, members and guests present were 16.

As the meeting was called to order, all reverently bowed their heads in remembrance of the departure from their midst of Mrs. August Rist, who had for the past third of a century prepared the superb and bounteous dinners for our Society.

After the excellent dinner was served by Mr. August Rist, the Society convened for its regular meeting. Drs. Glenn J. Smith and W. H. Pipes were elected members of the Bi-Parish Medical Society. Election of officers for 1943 resulted as follows:

Dr. Glenn J. Smith, President.

Dr. M. A. Walker, Vice-President.

Dr. E. M. Toler, Secretary-Treasurer.

Dr. C. S. Toler, Delegate to Louisiana State Medical Society.

Dr. E. M. Robards, Alternate Delegate.

The Scientific program was as follows:

Dr. T. S. Jones, Baton Rouge, La., read a paper on Trichomonas Vaginalis.

Dr. M. A. Walker, Angola, La., presented a paper on conditions at the hospital of the State Penitentiary on Health and Sanitation of the Institution, comparing conditions that existed when he assumed head duties, two years ago, which showed a vast improvement during his tenure of office. We trust that Dr. Walker will be retained and further improvement may develop from time to time, for the best interests of the state and the inmates of this institution.

A vote of thanks was extended to Drs. Jones and Walker for the presentation of their excellent and practical papers, also to Mr. August Rist for the superb entertainment and dinner prepared for us.

The Society adjourned to meet in the East Louisiana State Hospital at Jackson, La., the first Wednesday in March, 1943, at 7:30 p. m.

DR. W. J. ROBERTS, President.
DR. E. M. TOLER, Sec.-Treas.

JACKSON, LINCOLN, UNION PARISHES

At a meeting of the Jackson, Lincoln and Union Parishes Medical Society held at the Ruston Sanitarium, December 1, 1942, the following officers were elected for the year 1943:

Dr. J. B. Harris, Ruston, President; Dr. C. S. McDonald, Jonesboro, Vice-President from Jackson Parish; Dr. J. F. Tanner, Bernice, Vice-President from Union Parish; Dr. J. J. Bennett, Ruston, Secretary-Treasurer; Dr. C. C. Colvin, Bernice, Delegate.

DR. J. J. BENNETT,
Secretary-Treasurer.

THE INTERNATIONAL SOCIETY OF SURGERY

By a vote of the delegates from all the affiliated societies of the Americas, representing Argentina, Brazil, Canada, Cuba, Ecuador, Guatemala, Mexico, Paraguay, Peru, the United States, Uruguay, Venezuela, this oldest and most representative worldwide International organization (founded in 1902 through the initiative of the Belgian Surgical Society and the leading surgical societies of Europe and America) temporarily transferred its headquarters from Brussels, Belgium, to New York. The executive committee of the United States division, the largest, and most active contributor to its transactions, felt it their duty conjointly with their Latin-American colleagues, to rescue the society from the perils of the European conflagration, and especially of the Nazi domination.

By action of the Council of Delegates, the executive bureau of the society has been established in the Inter-American Division of the New York Academy of Medicine, directed by Dr. Mahlon Ashford, where Dr. Henry J. Cervantes, assistant secretary and treasurer, and editor of *America Clinica*, the official organ of the society for the Latin-Americas, will be able to render service to the fellows of the society and the medical visitors com-

ing from the Spanish and Portuguese-American countries.

The affairs of the society will be administered by an executive committee composed of Dr. Elliott C. Cutler, Col. M. R. C., U. S. A., chairman in absentia, Boston; Dr. Eugene Pool (Passed President Academy of Medicine, N. Y., and of the American College of Surgeons); Dr. Arthur W. Allen (Chairman Board of Regents American College of Surgeons), Boston; Rudolph Matas (Passed President International Society of Surgery, 1938, Passed President American College of Surgeons), New Orleans.

Dr. Jose Arce, dean of the University of Buenos Aires, is serving as acting president, in the absence of Professor L. Mayer, of Brussels, detained in Belgium by Nazi compulsion.

Until the restoration of peace and order in Europe, and when the war conditions will permit, the society will hold inter-American sessions coincidently with the annual Clinical Congress of the American College of Surgeons.

Dr. Matas, as acting secretary-treasurer of the executive committee, attended the meeting of Delegates and Fellows held in the New York Academy on November 12. Since his return he expressed his satisfaction with the perfect success of the transplantation and reorganization of the society, which was ratified by an act, witnessed by a very distinguished group of the New York Fellows and their Latin-American colleagues.

NEWS ITEMS

The Association of Military Surgeons of the United States, at a luncheon given December 15, 1942, in Washington, D. C., awarded the Gorgas Medal, founded by John Wyeth and Brother, Philadelphia, to Rear Admiral Edward Rodes Stitt, U. S. N., Brigadier-General Jefferson Randolph Kean, U. S. A., and Brigadier-General Frederick Fuller Russell, U. S. A., for distinguished service in Preventive Medicine for Our Armed Forces.

The fifth Annual Congress on Industrial Health this year will be held at the Palmer House in Chicago, January 11-13. A most interesting program has been provided for the three day session. Those interested in industrial health in medicine might well make it a particular point to attend this outstanding meeting.

A joint Committee of the Council on Foods and Nutrition of the American Medical Association and of the Food and Nutrition Board of the National Research Council has published a small booklet entitled "Food Charts: Foods as Sources of the Dietary Essentials." This small booklet can be obtained from the American Medical Association for the price of ten cents. The brochure should be of very great interest to those who are concerned with the problems of nutrition.

Dr. George W. McCoy, Director of Public Health of the Louisiana State University School of Medicine, attended the meeting of the National Foundation for Infantile Paralysis at New York City on December 3, 1942. Dr. McCoy is a member of the Medical Advisory Committee of the National Foundation.

First Lieutenant J. C. Swartzwelder, of the United States Army Medical Corps, formerly Assistant Professor in the Department of Public Health of the Louisiana State University School of Medicine, has been transferred from the LaGarde General Hospital to the 4th Service Command Laboratory at Fort McPherson, Atlanta, Georgia.

The date for the 17th annual meeting of the National Conference on Medical Services has been set for Sunday, February 14. This meeting is held annually in connection with the Congress on Medical Education and Licensure, which is scheduled this year for February 15 and 16. The place of meeting will be the Palmer House, Chicago.

The regular meeting of the Touro Staff was held on Wednesday, December 9 at 8 p. m. There was a clinico-pathologic conference with a discussion by Dr. Arthur Caire. Dr. Howard Mahorner reported on the "Control of Raynaud's Syndrome by Repeated Sympathetic Block." Drs. D. N. Silverman, Friedman, and J. D. Rives discussed "Fibrosis of the Stomach."

The National Society for the Prevention of Blindness announces that a prize of $250 will be awarded for the most valuable original paper during 1943 adding to the existing knowledge about the diagnosis of early glaucoma. The award will be made by the society with the guidance of an ophthalmological committee composed of Dr. Arnold Knapp, Dr. Manuel Uribe Troncoso and Dr. Mark J. Schoenberg.

SOUTHERN MEDICAL

The full list of the newly elected officers of the Southern Medical Association is given below:

President-Elect: Dr. W. T. Wootton, Hot Springs National Park, Ark.

Vice-President: Dr. Wyndham B. Blanton, Richmond, Va.

Chairman of Board of Trustees: Dr. Frank K. Boland, Atlanta, Ga.

Chairman of Council: Dr. Lucien A. LeDoux, New Orleans, La.

Executive Committee of Council: Dr. Lucien A. LeDoux, Chairman, New Orleans, La.; Dr. Oscar B. Hunter, Washington, D. C., and Dr. Curtice Rosser, Dallas, Tex.

UNITED STATES PUBLIC HEALTH SERVICE

Past Asst. Surgeon W. J. Fitzgerald has been relieved from duty in New Orleans and assigned to

the U. S. Merchant Marine Cadet Basic School, Pass Christian, Miss. Asst. Surgeon Charles N. Fiore and Asst. Surgeon Abraham Jacobs have been ordered to the Coast Guard Headquarters, Eighth Naval District, New Orleans.

AMERICAN COLLEGE OF PHYSICIANS

The Board of Regents of the American College of Physicians has announced the cancellation of their 1943 Annual Session, which was schedu'ed to be held in Philadelphia, April 13-16, 1943. This action was taken after thoughtful consideration of all factors involved, including an intimation from the Secretary of War and the Office of Transportation that larger national medical groups should not plan meetings at the time set; a growing difficulty in getting speakers and clinicians of top rank to maintain the usual standards of the program; prospect of greatly reduced attendance, because civilian doctors are faced with too great a burden of teaching and practice already; a decreasing active membership, due to approximately 25 per cent of all doctors being called to active military service. President James E. Paullin announced, however, that all other activities of the College would be pursued with even greater zeal, and that the College would especially promote regional meetings over the country and organize postgraduate seminars in the various military hospitals for doctors in the Armed Forces.

ANNOUNCEMENT FOR STATE MEDICAL JOURNALS

An intensive educational program on rheumatic fever and heart disease has been launched by the Metropolitan Life Insurance Company with the cooperation and advice of the American Heart Association, the Academy of Pediatrics, and the Crippled Children's Division of the U. S. Children's Bureau.

The purpose of this activity is twofold. Through popular literature, addresses, radio programs, news releases, and advertisements the public will be acquainted with the newer knowledge of rheumatic fever and rheumatic heart disease and their special war-time significance in overcrowded and insanitary environments. The program will stress the importance of early diagnosis, proper care for rheumatic fever victims, and the declining mortality rate among children in the younger age groups. The Metropolitan is using the services of its Field Staff in some three thousand communities, and it expects to reach over a million homes.

An equally important objective of this program will be to distribute to the medical profession up-to-date literature covering various aspects of the rheumatic fever problem. The printed materials for physicians will include a clinical booklet which sets forth in concise form the modern concept of the disease, criteria for its early diagnosis and treatment, methods of managing the disease and recognizing its cardiac involvements. This booklet is being developed in collaboration with special committees of the American Heart Association and the Academy of Pediatrics. The membership of these committees include: Drs. T. Duckett Jones and Edward F. Bland of Boston; Arthur C. DeGraff, Ann G. Kuttner, Homer Swift, and A. T. Martin of New York City; Ethel C. Dunham and O. F. Hedley of Washington, D. C.; Hugh McCulloch, St. Louis; and Stanley Gibson of Chicago. Others being consulted include Dr. May G. Wilson, New York City; Dr. Albert Kaiser, Rochester; Dr. David Rutstein, Albany; and Dr. Elizabeth Huse, Washington, D. C.

This program marks the first time that a national educational campaign has been undertaken to reach the general public and the medical profession with rheumatic fever literature, especially prepared according to the needs and interests of each group.

GASOLINE RATIONING

The following memorandum was distributed by the Tarrant County Medical Society of Texas. It has been very generally distributed not only to the local community doctors, but throughout the State of Texas. It is felt worth while calling this bulletin to the attention of Louisiana physicians.

HELPFUL HINTS FOR DOCTORS

Because physicians' services are a public necessity we doctors are to be furnished sufficient gasoline and tires to carry on the various activities of our profession. We are not a "privileged" class. Each and every one must regard the possession of a "C" card as a sacred trust. Let us not forget this for a single minute. Actually and literally you must keep your professional car with you. If the car goes out—you go with it.

When you apply for your "B" or "C" card be sure and list any and all uses to which it is to be put on your request blank. If you ask for additional supplies for your second car be sure that you can control its use. You will be held personally responsible for it same as your professional car.

IF YOU HAVE TWO CARS

Your personal professional car can only be driven 90 miles on non-professional use. If your spare car (other car) has a "B" or "C" card it can be driven only 90 miles for "non-professional" use. If it has an "A" card the whole 240 miles can be driven as you, your wife, or your family see fit.

If your wife drives you to the office in a car bearing a "B" or "C" card the whole trip must be counted part of your 90 miles of non-professional driving. Your car is in non-professional use when driven by anyone but the doctor—an exception being made to a nurse or assistant performing necessary and emergency mission for the doctor.

IF YOU HAVE ONLY ONE CAR

Remember this—if you have only one car you (or anyone else) can only use it for 90 miles of non-professional driving per month if it carries either a "B" or "C" card.

Stay with your car; keep it in your personal possession. Be very sure that your car is not driven while you are at the office. It is not possible for your wife to drive you to the office and then use the car while you work.

It may be hard to break habits that we and our families have developed but you will see that we as no other group will be watched closely by the public. Our Medical Society simply cannot allow any member to bring down suspicion on all the rest of us.

We are on our honor and we are patriotic. We have a job to do and we are going to do it—bravely—courageously—honestly.

Your committee,

Tarrant County Medical Society.

DEPARTMENT OF HEALTH

It is not generally realized to what extent the Laboratory Division of the Louisiana State Board of Health is utilized by the doctors in the State. In the year 1941 the Division examined over 600,000 specimens; in the present year it is estimated that there will be over 1,000,000 specimens sent to the laboratories for diagnostic purposes.

The United States Public Health Service evaluation survey of the Kolmer and Kahn modifications of the Wassermann test shows that the state laboratories rank extremely high in the results obtained in these tests, the presence or absence of syphilis.

AMERICAN ASSOCIATION FOR THE STUDY OF GOITER

Announcement that the Van Meter Prize would be awarded in 1942 was made on September 10, 1941, and after the Council had decided to call off the 1942 meeting, they decided that the Award should still be made provided the essays were of sufficient merit. Several essays were submitted and the special committee has made its report. The prize goes to Drs. Walter Mann and Charles P. Leblond, from the Department of Radiology and Anatomy of the University of Rochester School of Medicine and Dentistry, Rochester, New York. The title of their essay is "Chemical Transformation of the Iodine Fixed by the Thyroid Gland."

The next two essays received honorable mention, "The Nature of the Disordered Calcium and Phosphorus Metabolism in Thyrotoxicosis and Myxedema" by Dr. John W. Robertson; and, "Liver Changes Occurring in Hyperthyroidism with Special Reference to the Deleterious Effects of Anoxia" by Drs. Munro A. McIver and Eleanor A. Winter.

THE CRITICAL ANTIMALARIAL PROBLEM AND ITS SOLUTION

It is axiomatic that troops cannot operate successfully in endemic malarial areas without an effective antimalarial drug. Malaria is present throughout most of the tropical and sub-tropical world. The extent of operations in these regions is steadily increasing and it is conceivable that they may grow to tremendous proportions within a short period of time.

Ninety per cent of the world's customary sources of quinine was cut off when the Japanese invaded Java and the Philippines. India has in the past frequently imported cinchona bark from Java to bolster her supply. South America has for many years been importing bark from Java for domestic use.

What measures should be taken to safeguard our limited stocks? Early in May the War Production Board recognized the critical nature of the problem. In order to avail itself of medical advice it asked the National Research Council to form a Committee on Drugs and Medical Supplies to investigate and advise not only on the many problems which the quinine scarcity presented, but also on any other matters pertaining to Drugs and Medical Supplies which concerned the War Production Board. This committee has two subcommittees, one on Essential Drugs, the other on Hospital and Surgical Supplies. The Committee has also used the advice of other National Research Council Committees, such as the Subcommittee on Tropical Diseases for answers to specific problems in the therapy of malaria, and the Committee on Surgery for technical problems related to surgical supplies.

The program worked out by the joint efforts of the War Production Board and the National Research Council progressed as follows:

1. The War Production Board issued "Quinine Order M-131" which froze present stocks and restricted the use of quinine and other cinchona alkaloids to the treatment of malaria, except that quinidine could also be used for the treatment of cardiac disorders. Requests for quinine for the treatment of various other conditions including quinine urethane and quinine urea as local anesthetics, quinine in sodium morrhuate for varicose vein injections, quinine salts for induction of labor, quinine salts in the treatment of night cramps, multiple sclerosis, amyotrophic lateral sclerosis, myotonia atrophica, paralysis agitans and myotonia congenita, have all been carefully considered and denied with the exception of the use of quinine in the treatment of myotonia congenita.

2. Repeated persuasive efforts in the trade press have been instituted by the War Production Board to bring in all unopened packages of quinine, and now the machinery is being set up for bringing in opened packages, analyzing each and pooling according to the type of preparation.

3. To effect the maximum antimalarial value of available cinchona bark from South America, the National Research Council undertook to investigate the feasibility of substituting totaquine for quinine in the treatment of malaria. Totaquine had been recognized previously by the U. S. Pharmacopoeia, and a standard set which appears in the 12th Edition. This standard seemed to allow for too much latitude both in quinine content and in total crystallizable alkaloid content. Accordingly, the National Research Council asked the U. S. Pharmacopoeia to revise the monograph to obtain a more uniform standard. As the result the U. S. Pharmacopoeia adopted the following monograph which will appear in a supplement of the 12th Edition.

"Totaquine is a mixture of alkaloids from the bark of Cinchona Succirubra Pavon and other suitable species of Cinchona. It contains not less than 7 per cent and not more than 12 per cent of anhydrous quinine, and a total of not less than 70 per cent and not more than 80 per cent of the anhydrous crystallizable cinchona alkaloids, the designation "crystallizable alkaloids" referring to cinchonidine, cinchonine, quinidine, and quinine.

"Totaquine of a higher quinine percentage may be reduced to the official quinine standard by a mixture with Totaquine of a lower percentage, or with any of the diluents permitted for powdered extracts under Extracta, page 174, provided the total anhydrous crystallizable cinchona alkaloids do not fall below the required percentage.

"The National Research Council recommended the dose to be 10 grains three times a day for 7 days."

It was the considered opinion of the Subcommittee on Tropical Diseases, and the Committee on Medicine of the National Research Council, that in this dosage totaquine should prove to be as effective as quinine sulfate in the oral treatment of malaria. A satisfactory price structure has been set up for cinchona bark from South America by the Board of Economic Warfare, and every effort is being made to stimulate importation.

4. The production of atabrine has been stimulated by the War Production Board, and the present outlook on production sufficient to supply all anticipated needs is practically assured. Extensive chemical, pharmacological and clinical investigations have been conducted by various groups of the National Research Council working under grants by the Office of Scientific Research and Development. The chemical studies have adequately demonstrated the chemical purity of the atabrine produced in this country. Pharmacological and clinical investigations have revealed temporary gastrointestinal disturbances in a variable percentage of persons receiving atabrine in the suppressive (prophylactic) treatment of malaria. Further study is under way to elucidate the cause and remove if possible these disturbances. There is a tendency to the development of a yellow pigmentation of the skin during administration which disappears after the drug is stopped. This is harmless and not associated with any disturbance in liver function.

5. Experience with the use of atabrine by the British both therapeutically and as a suppressive, indicates that it is an effective antimalarial and in some ways superior to quinine. It is slower in action, and therefore not as useful in the initial treatment of malaria, but after one to three days of quinine therapy atabrine is exceedingly effective. At a recent meeting of the Subcommittee on Tropical Diseases of the National Research Council, the following program of dosage was endorsed as an efficient routine of therapy:

(1) *Combined QAP treatment.* (Method of choice.)

(a) *Totaquine or quinine sulphate,* 0.64 gram (10 grains) three times daily after meals for 2 or 3 days, until pyrexia is controlled. Then give

(b) *Atabrine,* 0.1 gram (1½ grains) three times daily after meals for 5 days. Then after 2 days without antimalarial medication give

(c) *Plasmochin,* 0.01 gram (3/20 grain) three times daily after meals for 5 days, except for the debilitated patient, who should receive only two doses daily. (Discontinue if toxic symptoms occur. Never give atabrine and plasmochin concurrently.)

(2) *Atabrine-plasmochin treatment.* (May be used for simple vivax infections and in other infections when no totaquine or quinine is available.)

(a) *Atabrine,* as above for 7 days. Then, after 2 days without antimalarial medication, give plasmochin, 0.01 gram three times daily for 5 days, as above.

(3) *Totaquine or quinine-plasmochin treatment.* (Method when no atabrine is available.)

(a) *Totaquine or quinine sulphate,* as above, for 7 days, during the last 5 of which accompany each dose of totaquine or quinine with plasmochin, 0.01 gram three times daily.

(4) *Suppressive treatment.* a. *Atabrine.* Give 0.1 gram (1½ grains) twice daily after meals on two days a week, allowing a 2 or 3 day interval between days of medication.

It was the consensus of this Subcommittee that until we have had more experience with the use of atabrine it should be used only under the guidance of a physician or public health officer.

6. Government stockpiles have been recommended not only for quinine but the other alkaloids, (cinchonine, cinchonidine and quinidine) and totaquine, and the Defense Supplies Corporation is purchasing against these stockpile recommendations.

It is anticipated on the basis of recent investigations by the Board of Economic Warfare that the barks from South America of low quinine content but sufficiently rich in total crystallizable alkaloids (quinine, quinidine, cinchonine, cinchonidine) to make totaquine of U. S. P. standards, will be found in sufficient quantity to enable totaquine to replace civilian quinine requirements. The amount of this bark which is available has been an unknown fac-

tor, because its low quinine content has not made it previously marketable.

If every physician in civilian practice and every public health officer will follow the recommendations of the Subcommittee on Tropical Diseases regarding the use of atabrine and will use totaquine in place of quinine whenever totaquine is available, an important and material conservation in our limited stocks of quinine will be accomplished.

Lewis H. Weed, M. D.

Chairman, Division of Medical Sciences
National Research Council

UROLOGY AWARD

The American Urological Association offers an annual award "not to exceed $500" for an essay (or essays) on the result of some specific clinical or laboratory research in Urology. The amount of the prize is based on the merits of the work presented, and if the Committee on Scientific Research deem none of the offerings worthy, no award will be made. Competitors shall be limited to residents in urology in recognized hospitals and to urologists who have been in such specific practice for not more than five years.

The selected essay (or essays) will appear on the program of the forthcoming meeting of the American Urological Association, May 31-June 3, 1943, Hotel Jefferson, St. Louis, Missouri.

Essays must be in the hands of the secretary, Dr. Thomas D. Moore, 899 Madison Avenue, Memphis, Tennessee, on or before March 1, 1943.

The Bureau of the Census, Department of Commerce, reports that there were 124 deaths in the City of New Orleans for the week which ended November 14, divided 78 white and 46 negro, with eight of these deaths occurring in children under one year of age. There was quite a considerable increase in the number of deaths the next week which ended November 21, there being 146 deaths reported in the city, 78 white, 68 colored, and 21 in small babies. The week finishing November 28 found 136 deaths occurring in the city, 81 of which were white people and 55 in the negro race, with 11 infantile deaths. There was quite a sharp increase in the number of deaths in the city in the week which closed December 5. The 164 deaths were divided 108 white and 56 negro. The increase in this week can be largely attributed to the deaths in the white population, as the negro number of deaths was stationary. Fifteen of the 164 deaths were infants under one year of age. It is interesting to note that so far this year there were over 404 less deaths in the City of New Orleans than last year, which in turn was an extremely healthy year. This same state of affairs exists all over the country. The death rate and the morbidity rate throughout the United States, fortunately for our great country, are smaller than they have ever been before in the history of the country.

INFECTIOUS DISEASES IN LOUISIANA

The Section of Epidemiology of the Louisiana State Board of Health shows that for the week ending November 14 there were reported the following diseases in numbers greater than 10: 238 cases of syphilis, 98 of gonorrhea, 27 of pulmonary tuberculosis, 18 of chanchroid, 12 of diphtheria, and 10 of scarlet fever. This is really a very remarkable report, for if the venereal infections are excluded, only three diseases occurred in this state in figures of 10 or more. During this week there were two cases of malaria reported, three of typhus fever, and one of meningitis. The week which terminated on November 21 finds 364 cases of syphilis listed, 195 of gonorrhea, 44 of pulmonary tuberculosis, 13 of diphtheria, 11 of mumps, and 10 of influenza. Jackson Parish this week reported nine cases of malaria, and there were two cases of typhus fever reported, one from Acadia and one from Caddo Parish. Nearly all of the cases of gonorrhea were reported from army camps, but only 51 of the 364 cases of syphilis. For the week which closed November 28 syphilis had risen to 342, gonorrhea 136, pulmonary tuberculosis 23, and chanchroid 15. This is even a more remarkable morbidity report than that of two weeks previous, because tuberculosis alone was the only non-venereal infectious disease which was reported in which there were more than 10 cases. No patients with malaria were found in the state that week, only one case of typhoid fever, and none of the unusual diseases. For the week which came to an end December 5, there were listed 340 cases of syphilis, 185 of gonorrhea, 23 of pulmonary tuberculosis, 17 of mumps, 13 of chanchroid, and 12 of food poisoning. There were six cases of typhus fever reported this week, and two of meningococcus meningitis. There were only three cases of typhoid fever in the state.

THE NEW DRUGS OF THE U. S. P. XII

Since the last Supplement to the U. S. P. XI was issued certain drugs have been adopted by interim revision in order to meet an immediate need for such official recognition. These drugs, although in the U. S. P. XII, are not listed as additions, inasmuch as technically they are already official. They are as follows:

Aleovitamin A and D; Insulin Injection; Oil of Cedar Leaf; Persic Oil.

Additions to the U. S. P. XII:

The very extensive list of new monographs in the U. S. P. XII is due to the addition of a rather comprehensive list of therapeutic substances as well as the new policy of recognizing some suitable dosage form for each therapeutically useful drug. An abbreviated list of only those substances classified as new drugs follows:

Acidum Aminoaceticum; Aethylis Carbamas (Urethane); Amaranthum; Amyleni Hydras (Tertiary Amyl Alcohol); Aqua pro Injectione; Bentonitum ("Wilkinite"); Butacainae Sulfas; Butylis Aminobenzoas ("Butesin," "Benzocaine," "Anesthesin"); Calcii Mandelas ("Camdelate Tablets"); Calx Sodica; Carbarsonum; Carbasus Absorbens (Gauze, Plain Gauze, Non-sterilized Absorbent Gauze); Carbasus Absorbens Adhaesivus (Adhesive Absorbent Compress); Carbasus Absorbens Sterilis (Sterile Gauze); Chloroazodinum ("Azochloramide"); Chorda Serica Chirurgicalis (Silk Sutures); Chorda Serica Chirurgicalis Sterilis (Sterile Silk Sutures); Dihydromorphinoni Hydrochloridum ("Dilaudid"); Diphenylhydantoninum Sodicum ("Dilantin Sodium"); Emplastrum Adhaesivum Sterile (Sterile Adhesive Tape); Ergonovinae Maleas ("Ergotrate Tablets," Ergometrine, "Basergin Tablets"); Ergotaminae Tartras ("Gynergen"); Estradiolis Benzoas ("Progynon-B," "Dimenformon Benzoate"); Estronum (Theelin); Eucatropinae Hydrochloridum ("Euphthalmine Hydrochloride"); Extractum Perpolitionum Oryzae (Extract of Tikitiki, Extract of Rice Bran, Extracto de Salvado); Ferri Sulfas Exsiccatus; Gelatum Alumini Hydroxidi (Colloidal Aluminum Hydroxide) ("Amphojel," Creamalin"); Gelatum Alumini Hydroxidi Siccum ("Amphojel Tablets"); Globulinum Immune Humanum (Measles Prophylactic, Placental Extract); Glycerylis Triacetas (Triacetin); Hexylresorcinol ("Crystoids Anthelmintic"); Injectio Digitalis (Digitalis Ampuls); Injectio Mercurophyllinae ("Mercupurin"); Ligamentum Carbasi Absorbens (Roller Gauze Bandage); Liquor Chloridorum Trium Isotonicus (Ringer's Solution); Liquor Chloroazodini (Azochloramide in Triacetin); Liquor Iodi; Liquor Sodii Citratis Anticoagulans; Liquor Tribromoaethanolis (Solution of Tribromoethyl Alcohol); Magnesii Trisilicas; Menadionum (2-Methyl-naphthoquinone; 2-Methyl-1, 4-napthoquinone) ("Kayquinone," "Proklot," "Thyloquinone"); Mersalyl ("Injectio Mersalylis et Theophyllinae"); Nebula Epinephrinae Hydrochloridi; Neostigminae Bromidum; Neostigminae Methylsulfas ("Prostigmine Bromide," "Prostigmine Methylsulfate"); Nicotinamidum (Nicotinic Acid Amide, Niacinamide; Oleum Hippoglossi; Ouabainum (G-Strophanthin); Pamaquinae Naphthoas (Aminoquin Naphthoate) ("Plasmochin"); Perpolitiones Oryzae (Rice Polishings); Plasma Humanum Normale Citratum (Normal Human Plasma); Quinacrinae Hydrochloridum (Mepacrine Hydrochloride) ("Atabrine"); Riboflavinum (Lactoflavin, Vitamin B_2, Vitamin G); Serum Humanum Normale (Human Serum); Serum Immune Morbillosi Humanum (Measles Convalescent Serum); Serum Immune Scarlatinae Humanum (Scarlet Fever Convalescent Serum); Sulfapyridinum; Sulfapyridinum Sodicum Sterile; Sulfarsphenamina; Sulfathiazolum; Sulfobromophthaleinum Sodicum ("Bromsulphalein Soluble"); Syrupus Glycyrrhizae; Tetracainae Hydrochloridum ("Pontocaine Hydrochloride"); Tetrachloroaethylenum (Perchloroethylene, Ethylene Tetrachloride) ("Nema"); Theobromina et Sodii Acetas ("Agurin"); Totaquina ("Quinium Quinetum"); Toxoidum Tetanicum; Tribromoaethanol ("Avertin Crystals"); Trichloroaethylenum; Unguentum Aethylis Aminobenzoatis; Urea (Carbamide).

WOMAN'S AUXILIARY

Louisiana State Medical Society

This month we are featuring the activities of two of our North Louisiana Auxiliaries, Caddo and Ouachita. Both are busy groups, doing worthwhile things as you may judge from the following:

Mrs. J. M. Bodenheimer of Shreveport tells that the Caddo programs this year are being prepared and presented by Auxiliary members instead of outside talent. In October "We Are On the Air" was the subject given, and it outlined the activities of the Auxiliary since its inception 14 years ago. In November the Program Committee presented "The Medical Auxiliary in War Work," which showed that practically every member is engaged in some form of War work. That is a splendid showing. Special honor was paid to their men in Service at this meeting, too.

This month the Shreveport Medical Society and the Auxiliary are giving a Christmas party for soldiers at the U. S. O., instead of the usual party they have for themselves at this time. That is a fine thing to do, don't you think? And the group is continuing their educational work through the sale of Tuberculosis Seals and Hygeia. They sponsor a Toy Loan Library at Charity Hospital.

Mrs. A. G. McHenry of Monroe reports from the Ouachita Auxiliary. In November the State President, Mrs. Clarence B. Erickson, was the guest speaker at the business session and afterwards was tendered a beautiful luncheon by the members.

In December Dr. Charles R. Gowan of Shreveport gave an interesting and informative talk on Progress made in Tuberculosis Work. Excellent reports were given by the chairmen of Hygeia, Red Cross and Christmas Seal Sale. Mrs. W. C. Simpson reviewed an article from the Bulletin.

The Auxiliary voted to cooperate with the Christmas present program of the U. S. O., and Mrs. A. G. McHenry, Mrs. G. B. Wilkins and Mrs. R. E. Stevison were appointed to serve on the committee to help with that work. Another group composed

of Mrs. E. G. Calvert, Mrs. H. V. Collins and Mrs. C. P. Gray was appointed to decorate a Christmas Tree at Conway Hospital.

Eighteen members and Dr. and Mrs. Charles Gowan of Shreveport, guests, attended this very fine meeting.

Thank you very much, Caddo and Ouachita, for telling us what you are doing. You inspire the rest of us to greater efforts.

And now, may 1943 bring a brighter day to our distressed world. And may we, as Auxiliary Members be given greater strength and greater will to do a greater part in hastening that day of Peace.

MRS. JAMES W. WARREN, Chairman,
Press and Publicity.

—o—

BOOK REVIEWS

Intestinal Obstruction: By Owen H. Wagensteen, B. A., M. D., Ph. D. 2d ed. Springfield, Ill., Chas. C. Thomas Co., 1942. Pp. 484, illus. Price $7.00.

Published in its second edition the author has endeavored to bring the subject of intestinal obstruction up to date in this work. In reviewing this book one can readily see that the literature has been well considered and all of the worth while data included.

Many major additions have been inserted since the first edition in 1936, and as stated by the author in the preface: "The plan of this edition is more natural and the scheme employed for the discussion of the subject is under four distinct sections which are: (1) Effects of Obstruction; (2) General Diagnostic Considerations in Recognition of Bowel Obstruction; (3) General Therapeudic Considerations in Its Management; (4) Special Obstructions." There are tweny-five chapters and 143 figures. The references are systematically arranged at the end of each chapter.

The subjects discussed are many, such as the importance of swallowed air in distention, the use of the Miller-Abbott tube, closed aseptic resection, and the use of sulfonamides in treatment of peritonitis. The author has also stressed the idea of linking the clinical with the x-ray evidence in the diagnosis of obstruction. One who wishes to practice modern medicine or surgery should be acquainted with this work.

EMILE BLOCH, M. D.

Methods of Treatment in Postencephalitic Parkinsonism: By Henry D. von Witzleben. New York, Grune & Stratton, 1942. Pp. 164. Price, $2.75.

This well arranged volume dealing with the methods of treatment in postencephalitic Parkinsonism with preface by Theodore J. C. von Storch is one of great interest and instruction to our present therapy of illnesses mentioned therein.

The volume is substantially bound and in turn there are interpretations and explanations of the twelve chapters following the introduction and preface. It is interesting to note the evaluation of the various forms of treatment given to the originators of these treatments; likewise, the recognition of experimentation done in the various fields of pharmacology, chemistry and biochemistry referable to the illnesses mentioned.

It is interesting also to read of the various forms and types of therapy that have been used in times past in attempting a cure or administering a measure for the relief of one of the most uncomfortable series of illnesses met within mankind.

The bibliography, complete in all details and up-to-date, would display the fact of the painstaking efforts on the part of the author to incorporate for our benefit all minute medical information which would aid us in making life pleasanter for the patient.

The book is easy to read, printing is standard and clear and should be used as a vade mecum for the general practitioner as well as the neuropsychiatrist.

The volume is affectionately dedicated to the memory of the late Constantin Baron Von Economo and to Felix Stern.

WALTER J. OTIS, M. D.

Medical State and National Board Summary: By William H. Kupper, M. D., with a foreword by Earl S. Hallinger, M. D. Patterson, New Jersey, The Colt Press, 1942. Pp. 369. Price, $4.50.

In this small volume the author has made available a great deal of practical information pertaining to the fundamental principles of the science of medicine which ordinarily occupy many much larger volumes. In the preface the statement is made that "this condensation has been made possible by means of illustrations, by eliminating repetitions and by the use of a compact style of presentation."

The chapters on anatomy and biochemistry are illustrated by easily interpreted drawings and diagrams, and at the end of each of the eleven chapters on pre-clinical and clinical subjects two pages are devoted to questions and answers. Normal and disease conditions are described under each heading, together with methods and special technics in diagnosis. Treatment of the more common disorders is mentioned briefly.

Several pages are devoted to National Board questions and answers. Three pages summarize briefly the history of medicine.

The chapter on National Board questions alone will prove "an oasis in the desert" to the bewildered and perplexed state or national board applicant. The reviewer would highly recommend Dr. Krupper's book to the physician in general practice, to the specialist who confines much of his reading to his own specialty and to the medical student in need of a handy, inexpensive summary of information on subjects that confront him daily.

ELIZABETH BASS, M. D.

Stedman's Practical Medical Dictionary: By Stanley Thomas Garber, B. S., M. D. 15th ed. rev. Baltimore, Williams & Wilkins Co., 1942. Pp. 1257. Price, $7.50.

Stedman's Medical Dictionary is too well known to the medical profession to need any complimentary or commendatory remarks. It has gone through fifteen editions and now is under the aegis of Dr. Stanley T. Garber. As a medical dictionary the volume is all that could be desired. The reviewer has used the dictionary for many years and it has never failed him. He has reviewed most flatteringly new editions for the last twenty years. This time the reviewer offers no criticism nor unfavorable comment on the dictionary as such but he does criticize vigorously the new font most emphatically. It is only with the greatest difficulty that the reader moves from the sharp black-faced type to a type which is very small not only in size but in thickness. It is truly almost a task to make use of the dictionary because of this change which the publishers or the editor seemed to think would be an advance in the art of typography.

J. H. MUSSER, M. D.

The Toxemias of Pregnancy: By William J. Dieckman, M. D. St. Louis, C. V. Mosby Company, 1941. Pp. 521. Illus. pl. Price $6.50.

This excellent monograph contains 500 pages, with 50 illustrations and three colored plates. It is divided into six sections. The first section of 56 pages considers the classification, incidence and pathology of the toxemias of pregnancy. The second section of 166 pages under the general heading "Normal and Abnormal Physiology" includes physiochemical determinations, blood pressure, renal physiology; the anatomy, physiology and pathology of the liver; the ocular system, the placenta, the endocrines and finally edema in pre-eclampsia and eclampsia. Section three contains 47 pages and considers the etiology of eclampsia and includes a discussion of those factors influencing eclampsia. Section four (89 pages) deals with the clinical aspects of the toxemias of pregnancy, and section five (88 pages) with treatment. The final section, six (52 pages), is devoted to maternal and fetal prognosis and prenatal care.

Dieckman has contributed extensively to the literature in the field of toxemias of pregnancy, having done a large amount of original research. This work includes not only the results of his own investigations but those of others, including frequent and liberal quotations. The author shows a mastering of the subject and a complete familiarity with the literature and investigations of others, and while much of the material is familiar to those who have followed recent literature it is well organized, thoroughly digested and ably presented.

The author states that he has a dual purpose in writing this book, first "to acquaint the obstetrician with some of the recent contributions on physiology pertaining to obstetrics" and secondly "to acquaint the investigator, untrained in obstetrics, with some of the physiology and pathology of obstetrics." He succeeds to a considerable degree in both of his aims. This book should be carefully read by everyone interested in obstetrics and the toxemias of pregnancy and kept at hand as a ready, valuable reference.

B. B. WEINSTEIN, M. D.

Directory of Medical Specialists Certified by American Boards: Compiled by the Advisory Board for Medical Specialties. New York, Columbia University Press, 1942. Pp. 2495. Price $7.00.

The second edition of the Directory of Medical Specialists does credit to those who compiled it. Any directory of this kind represents a tremendous amount of work and a detailed investigation. The editor has done his task well.

The directory can be recommended to the medical profession as a whole without reservation. It will prove of inestimable value to those who wish to refer patients to qualified specialists either in their own city or in cities elsewhere throughout the United States.

J. H. MUSSER, M. D.

PUBLICATIONS RECEIVED

J. B. Lippincott Company, Philadelphia: Fractures, by Paul B. Magnuson, M. D., F. A. C. S.

E. P. Dutton & Company, New York: Doctor Bard of Hyde Park, by John Brett Langstaff. Castor Oil and Quinine, by George Wonson Vandegrift, M. D.

W. B. Saunders Company, Philadelphia: Manual of Dermatology, Military Medical Manuals, issued under the Auspices of the Committee on Medicine of the Division of Medical Sciences of the National Research Council.

Charles C. Thomas, Springfield, Illinois: The Hemorrhagic Diseases and the Physiology of Hemostasis, by Armand J. Quick, Ph. D., M .D.

Columbia University Press, New York: A Venture in Public Health Integration, the 1941 Health Education Conference of the New York Academy of Medicine.